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A TEXT-BOOK

OF THE

THEORY AND PRACTICE

OF

MEDICINE.

BY

AMERICAN TEACHERS.

EDITED BY

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GENERAL CONSIDERATIONS CONCERNING THE BIOLOGY OF BACTERIA, INFECTION AND IMMUNITY.

BY WILLIAM H. WELCH.

THE parasites of human beings include members both of the animal and of the vegetable kingdoms. The animal parasites belong to the divisions protozoa, worms, and arthropods; the vegetable parasites, to the bacteria and fungi. The term pathogenic micro-organisms, according to common although somewhat arbitrary usage, includes all of the vegetable parasites and only the protozoa among the animal parasites.

The infectious diseases are caused by pathogenic micro-organisms. The special characters of the micro-organisms which cause the infectious diseases considered in this work are described in the articles treating of these diseases.

It is thought desirable to bring together in a separate article certain general considerations concerning infection. The most important advances in our knowledge of infectious diseases have come from the study of bacteria. Some knowledge of the general biology of bacteria is essential to an understanding of pathogenic bacterial species and of their relations to disease.

The most important protozoa at present known to be concerned in the causation of human diseases are the malarial plasmodium and the dysenteric amœba. These are described in connection with their respective diseases. An account of the general biology of protozoa, although of increasing interest and importance to human medicine, would not in the present imperfect state of our knowledge of this subject materially further the aim of this article.

Pathogenic fungi are of comparatively little importance to internal human medicine.

GENERAL BIOLOGY OF BACTERIA.

MORPHOLOGY AND CLASSIFICATION.

THE name "bacteria," signifying *staves*, is a convenient but etymologically incorrect one for the group of micro-organisms also called or included under schizomycetes or fission fungi, schizophytes, microphytes, protophytes, and, in common parlance, microbes or germs.

The chief reason for regarding bacteria as members of the vegetable kingdom is that they are believed to have more morphological and physiological properties in common with the simplest undoubted plants than with the lowest animals. They are more closely allied to the lower algæ called schizosporeæ or schizophyceæ than to the true fungi.

Bacteria are minute, achlorophyllous,[1] unicellular plants which multiply by repeated binary fission, and in many instances also by the formation of spores.

Nuclei have not been satisfactorily demonstrated in bacteria. Inasmuch as the staining reactions of the bacterial cell are more like those of nuclei than of protoplasm, it has been suggested that this cell is composed essentially of nuclear substance or of an intimate mixture of nuclear material and of protoplasm. Bütschli and P. Ernst regard as nuclei certain isolated nuclear-staining granules or larger bodies which are demonstrable in some bacteria. It is a growing belief that nuclear material is essential to the existence of a living cell capable of reproduction, so that it is probable that bacteria contain nuclear substance in some form.

The bacterial cell is believed to be always enveloped by a membrane. This membrane is considered by some to be an outer condensed layer of protoplasm, and by others to be the innermost layer of a gelatinous capsule which, at least under certain circumstances, envelops most if not all bacteria. Bacteria which have a readily demonstrable capsule capable of staining by dyes are described as capsulated.

The substance of the bacterial cells, composed, according to Nencki, essentially of an albuminous material called mycoprotein, appears usually uniform and colorless, but it may present various colors, as yellow, red, green, or violet. In some species the cell may contain starch; in the group of sulphur-producing bacteria, granules of sulphur; and in some of the higher forms, the cell-membrane may be stained various shades of brown by deposits of iron salts. The cell-substance, instead of being uniform, sometimes contains clear vacuoles, sometimes is finely or even coarsely granular, and sometimes presents parts or granules staining more deeply than the rest of the cell or of a different tint, and variously interpreted as condensed or retracted areas of protoplasm, as nuclei, as concerned in the formation of spores, and as degenerations. Probably these isolated, deeply-staining particles, which form important morphological criteria in some species of bacteria, are not all of the same character and significance.

The bacteria are divided, according to their forms, usually called growth-forms, into spheres or micrococci or cocci, rods or bacilli, and spirals or spirilla.

The cocci (see Fig. 1, A) are farther subdivided, according to their arrangement, into diplococci or paired cocci, streptococci or chain cocci (also torula), merismopedia or merista or tetrads or tetragonous cocci (arranged in tablets or in fours), sarcina or packet cocci (of eight or more cells), and ascococci or clumps of cocci enclosed in dense capsules. These differences in arrangement can be explained by the varying direction of the planes of division of the cells and by the separation or the juxtaposition of the cells after division.

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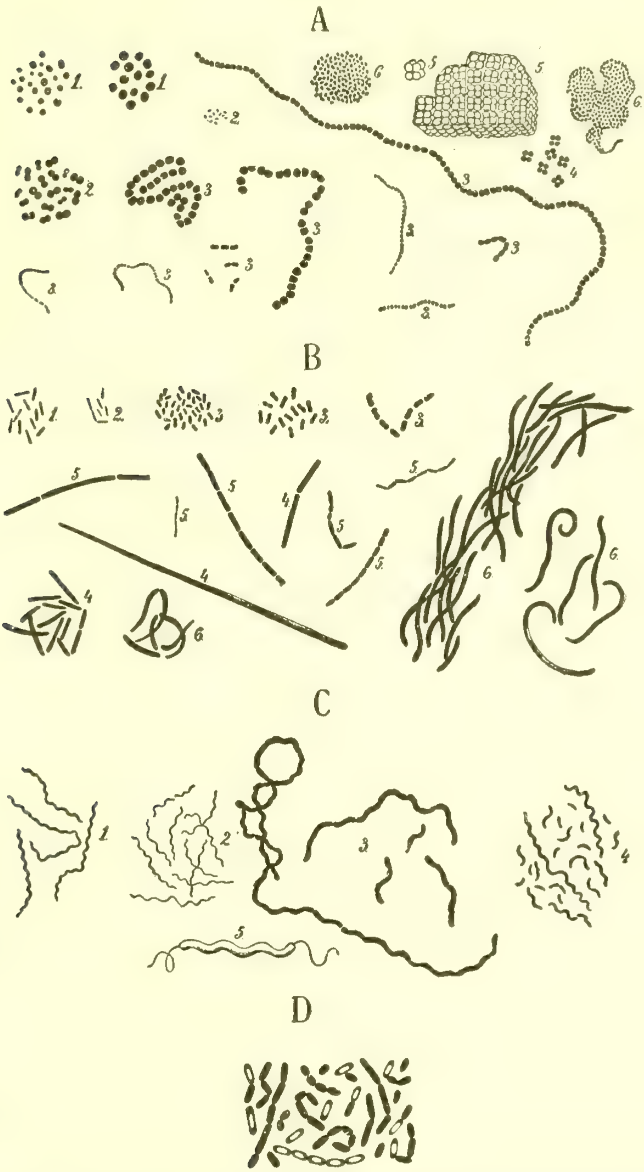
[1] Two or three species of bacteria have been described as containing a green coloring matter believed to be chlorophyl.

FIG. 1.



Forms of Bacteria: A, Micrococci; B, Bacilli; C, Spirilla; D, Bacilli containing spores, (Baumgarten): A^1, single, large and small cocci; A^2, large and small diplococci; A^3, streptococci of different sizes; A^4, micrococcus tetragenus; A^5, sarcina ventriculi; A^6, staphylococci; B^1, rather thick bacilli; B^2, slender bacilli; B^3, dividing bacilli—dumb bell or figure-of-8 shapes; B^4, long bacilli; B^5, bacilli in chains; B^6, curved and wavy bacilli; C^1, spirillum Obermeieri; C^2, spirochæte dentalis; C^3, Deneke's spirillum; C^4, spirillum choleræ Asiaticæ, (comma bacilli and spirilla); C^5, spirillum volutans (vibrio) with flagella; D, bacillus subtilis, containing spores, appearing as clear spaces. All (except A^5 and C^5) magnified 950 diameters.

All straight bacteria (see Fig. 1, B), in which one diameter is greater than the others, are called bacilli, with the exception of those cocci which may have assumed an oval or elliptical shape, usually just before division into spheres.

There are forms of bacteria on the border-line between rods and spheres, which some prefer to call cocci and others bacilli, but the tendency is to call even the very short oval forms bacilli, which were formerly often designated as cocci.

The old division of rod bacteria into bacteria proper, or short rods without formation of spores, and bacilli, or longer, spore-bearing rods, is now generally abandoned, although the name "bacterium" with a distinctive epithet, such as bacterium coli commune, is still often used to designate certain species. Bacteria in the form of long threads are often called leptothrix. Bacilli in chains are sometimes called strepto-bacilli. The name "clostridium" is applied to a spindle-shaped bacillus.

Cohn's division of spiral bacteria (see Fig. 1, C) into spirilla, or rigid, spirally-twisted forms; vibrios, with sinuous, low, less pronounced twists; and spirochæte, with long, flexible, corkscrew curves, is now generally abandoned, and the name "spirilla" is used for the whole group, although usage still often retains the terms "vibrio" and "spirochæte" with distinctive epithets, such as vibrio Metchnikovi and spirochæte dentalis, for certain species. Singly-curved bacilli are called comma bacilli. Many of these grow into regular spirilla, and they are therefore better designated as spirilla. The spirillum of Asiatic cholera is often called the cholera bacillus or the cholera vibrio.

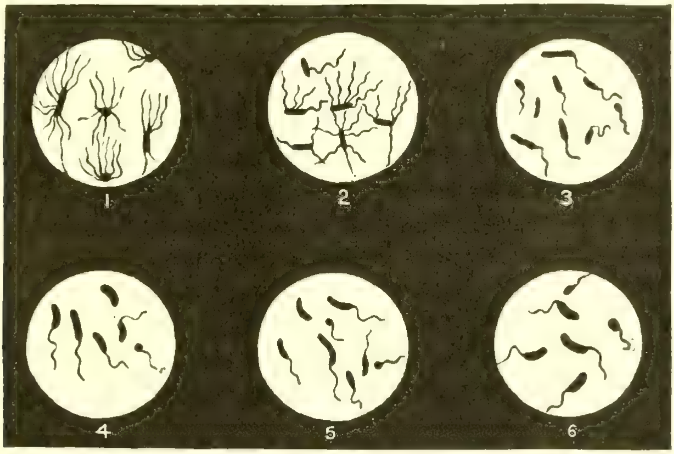
The botanists usually rank among the bacteria or schizomycetes certain forms which are larger and more highly differentiated than those already described, and which may show an appearance of branching, a distinction between base and ends of the filaments, and considerable variety of shapes even in one and the same species. Most of these, such as cladothrix, crenothrix, and beggiatoa, occur in water. Actinomyces (see Vol. I., Figs. 29 and 30) is believed to belong to the group of more highly organized bacteria. These highly differentiated forms are not, however, recognized as bacteria by all authorities, and certainly they should be separated from the other bacteria.

In old cultures and in unfavorable media many bacteria may present various anomalous shapes, such as swellings, varicosities, pear-shapes, twists, bends, and greatly elongated as well as abnormally short or disintegrated forms. These are spoken of as involution forms. Some of these forms may, however, be seen in vigorous fresh cultures of certain species, such as the bacillus diphtheriæ, and here it does not seem proper to regard them as involution or degeneration forms.

There has been much controversy as to constancy of form among bacteria. As regards this feature, bacteria may be classed as monomorphic, or constant in form; oligomorphic, or variable in form within relatively narrow limits; and pleomorphic, or capable of assuming the various growth-forms of rods, spirals, and spheres. Most of the pathogenic bacteria are monomorphic or oligomorphic; the bacillus proteus and many of the highly-organized bacteria are pleomorphic. By changes in environment, especially in the composition of culture-media, many bacteria present considerable diversity in form, but this diversity relates especially to variations in length and thickness and to the occurrence of involution forms.

Many bacilli and spirilla are motile, whereas only two or three species of cocci are known to be motile. This motility is due to the presence of delicate hair-like flexible processes called flagella, which can be stained by special methods, but otherwise, except with the larger spirilla, are usually invisible. There may be only a single flagellum at one end of the cell, or flagella at both ends, or flagella also along the sides. (See Fig. 2.)

FIG. 2.



Bacteria showing flagella: 1, bacillus of typhoid fever; 2, bacillus coli communis; 3, spirillum of Finkler and Prior; 4, spirillum of Deneke; 5, vibrio Metchnikovi; 6, spirillum of Asiatic cholera (Nicolle and Morax).

Many species of bacteria under certain definite conditions form spores. High temperature, free supply of oxygen, and exhaustion of the supply of nutriment are regarded as favorable to spore-formation, but, at least for many species, these conditions are not essential. Endogenous spore-formation (Fig. 1, D) is that mode which has been longest known and is best understood. It is not known to occur in cocci, is infrequent in spirilla, but is common in bacilli. There is first developed from the protoplasm of the cell a small granule which grows into a sharply circumscribed, highly refractive round or oval body, situated usually either in the middle or in one end of the cell, which it may cause to bulge at the site of formation, and from which it is set free by disintegration of the mother-cell. Only one spore is formed, as a rule, in a single bacterial cell. By the ordinary methods of staining bacteria the spore usually appears colorless, and in aqueous media refractive, but it can be stained by special methods. Spores are characterized by greater resistance to heat, desiccation, and other injurious influences than are the vegetative bacterial cells. They consist of an outer dense envelope called the exosporium, and an inner condensed protoplasmic and probably also nuclear material called the endosporium. Placed under suitable conditions, the spore germinates by an outgrowth of the endosporium, in some species through the ruptured ends, in others through the sides of the exosporium.

In distinction to endospores, arthrospores have been described by De Bary and Hueppe. These arthrospores are represented by entire bacterial cells which, it is claimed, have been transformed into bodies with the resisting properties of spores. Cocci, according to this view, may form arthrospores. De Bary and Hueppe have attempted to classify bacteria according to their mode of spore-formation, but this attempt has not met with much acceptance. In fact, the whole doctrine of arthrogenic formation of bacterial spores is obscure, and does not, at present, seem to rest upon any certain foundation.

Masses of bacteria held together by sticky intercellular substance are called zooglœa. Masses produced by the growth of bacteria upon solid media are called colonies, the distinctive characters of which form one of the most important means of differentiating bacterial species.

It is now recognized by biologists that a scientific systematic classification of plants and animals should be based as little as possible upon physiological differences or differences in function. Hence the higher plants and animals are now classified chiefly according to their structure and development. It is impossible with our present imperfect knowledge to furnish a systematic classification of bacteria based upon these morphological principles, and therefore analogous to the classification of the higher orders of plants and animals. We make as much use as possible, in attempting to classify bacteria, of morphological resemblances and differences, but we are obliged to take into account all of the other properties, including differences of function. The definition of any given bacterial species amounts, therefore, essentially to an enumeration of all of its known properties, such as its form, its origin, its staining reaction, its chemical products, its behavior in a variety of culture media and in the living body. The name given to the species is usually the name of the growth-form, such as micrococcus, bacillus, spirillum, with some distinctive epithet intended to indicate one or more characteristic properties, such as color-production, source, arrangement, power of causing fermentation or putrefaction, name of discoverer, cultural peculiarity, or, especially in the case of pathogenic bacteria, the disease caused by the bacterium; for example, staphylococcus pyogenes aureus, bacillus coli communis, micrococcus tetragenus, bacillus butyricus, bacillus saprogenes, spirillum Obermeieri, bacillus fluorescens liquefaciens, bacillus anthracis. Under these circumstances it is evident that what is described and supposed to be a species may subsequently prove to be a race or variety of some species, or, on the other hand, may correspond to a group of species. Notwithstanding these difficulties, and in spite of our inability to define bacterial species in terms analogous to those used for the higher plants and animals, it can scarcely be doubted that bacteria are divisible into distinct species just as much as are the higher organisms.

The present classification of bacteria therefore amounts to little more than a grouping of the many bacterial species under their growth-forms, micrococcus, bacillus, etc., with subdivisions relating to biological or chemical properties, such as power of liquefying gelatin, color-production, ability to bring about fermentation, pathogenic power, etc. The attempts which have been made to conform to the classification of the higher plants by grouping together in a table species of bacteria into genera and genera into families have not been thus far sufficiently successful or of enough practical utility to make it worth while to consider them here.

FOOD ; VITAL MANIFESTATIONS.

The capacity of rapid multiplication by division is one of the most striking properties of bacteria. Under favorable circumstances a bacterial cell has been known to divide into two within the space of twenty minutes. If multiplication at this rate proceeded unchecked, the progeny of a single bacterium would be enough in a few days to fill up all the oceans of the world. A check is soon put to this unlimited growth by lack of food, accumulation of bacterial products, and by various other influences, which will be hereafter described, hostile to the multiplication of bacteria.

The food of bacteria, like that of all living things, must contain carbon, nitrogen, oxygen, hydrogen, and certain mineral salts. In general, the most suitable food for bacteria is furnished by the diffusible albuminous substances and the carbohydrates, and these, therefore, constitute the chief ingredients of our artificial culture media. Bacteria, however, differ widely as to their demands for food. Some are capable of obtaining their nitrogen from ammonium salts, nitrites, and nitrates. Being destitute of chlorophyl, they are incapable (with possibly a few exceptions) of assimilating carbon dioxide, but many can obtain their carbon not only from starch and sugars, but also from many organic acids, glycerin, and other alcohols. Hydrogen and oxygen are obtained partly from the same sources as the nitrogen and carbon, and partly from water and free oxygen.

Many pathogenic bacteria require albuminous food, and some require this to be of such special quality that they will not grow in our artificial media, as, for example, the spirillum Obermeieri and the bacillus lepræ. At the other extreme are the so-called water bacteria, which Bolton found would multiply with the slight trace of organic material present in ordinary distilled water.

Most bacteria prefer an alkaline reaction of the medium in which they grow, but many will grow in neutral, not a few in acid media, and some even better in acid than in alkaline media.

A certain amount of moisture is essential for bacterial growth. In general, bacteria grow best when their food is not too concentrated.

The temperature limits within which bacteria may develop differ widely, not only for different species, but often also for the same species. The optimum temperature for most pathogenic bacteria is between 30° and 40° C. Usually bacterial growth ceases below 5° to 10° C. (for many pathogenic bacteria at a much higher point) and above 45° to 50° C., but bacteria have been described capable of some multiplication at freezing temperature, and others at temperatures between 60° and 70° C.

Many bacteria require free oxygen for their development, and are therefore called obligate aërobes; others will grow only in an atmosphere free from oxygen, and are called obligate anaërobes; and still others can develop either with or without free oxygen, and are called facultative anaërobes or facultative aërobes. Most pathogenic bacteria are facultative anaërobes; some are obligate anaërobes.

Bacteria by their growth in various media manifest functional activities, some of which are of little significance, but some are of the highest importance in the economy of nature, in the preparation of food, in the industries, in hygiene, and in pathology.

Bacteria are essential for the continuance of life on this globe by their work in decomposing dead vegetable and animal substances into water, carbon dioxide, and the simple nitrogen compounds which form the food of the higher plants and without which they would perish.

The processes of nitrification which take place on a vast scale in the soil are due to the activity of the so-called nitrifying bacteria, of which there are two kinds—one to decompose ammonia into water and nitrous acid, and the other to transform nitrous acid into nitric acid, the resulting nitrates forming the nitrogenous food of chlorophyllous plants. There are denitrifying bacteria which may undo the work of the nitrifying, and reduce nitrates to nitrites and ammonia. The nitrifying bacilli, although devoid of chlorophyl, are capable of forming organic compounds out of purely inorganic matters, and flourish best in media free from organic matter.

The so-called thiogenic bacteria transform sulphuretted hydrogen into sulphur and sulphuric acid.

Among the most important activities of bacteria are those concerned in various fermentations. These fermentative bacteria are called zymogenic bacteria. Those causing the particular kind of fermentation called putrefaction, in which albuminous and other highly organized nitrogenous substances are decomposed with stinking products, are called putrefactive or saprogenic bacteria.

The investigations of Schwann, Schulze, Cagniard-Latour, and, above all, Pasteur, which have led to the establishment of the doctrine that fermentation and putrefaction are due to living organisms, chiefly bacteria and fungi, occupy, together with those inaugurated by Spallanzani concerning spontaneous generation, a very important place in the historical development of the doctrine that the infectious diseases—which, indeed, have been called zymogenic in consequence of their analogies to fermentative processes—are also caused by living organisms.

The study of the various fermentations produced by bacteria and the yeast fungi is of much chemical interest and of great industrial importance, and has become a specialized department of bacteriology which has its own literature.

Here can be mentioned only, as an example of the more important fermentations produced by the yeast fungi, the alcoholic from sugars, and by the bacteria from carbohydrates, the alcoholic, the lactic acid, the butyric acid, the various slimy, viscous, or mucic fermentations, and the fermentation of cellulose with production of marsh gas; the fermentations of the alcohols, of which the most important is the acetic-acid fermentation of ethyl alcohol; the fermentations of the fatty acids; the fermentations converting milk into koumyss.

Each species of the zymogenic bacteria is endowed with the power of setting up its own special kind of fermentation, although a single species may be capable of causing different fermentations, or the same variety of fermentation may be produced by a number of species, or in some fermentations two or more species may be concerned simultaneously or consecutively. These fermentations are intimately associated with the vital processes of the organisms producing them, although it is not known in what way, and they cannot be carried to completion by any soluble ferments which have been isolated from the organisms.

Soluble ferments, called enzymes, are, however, produced by bacteria, and doubtless play an important rôle in the metabolism of these lowly organisms as well as in the higher plants and animals. The chemical action of these unorganized ferments is mainly hydrolytic, and does not bring about such complicated changes as do the living bacteria in setting up fermentations. Some of these unorganized ferments have been isolated from cultures of bacteria. Bacteria are capable of producing enzymes with the properties of diastase, of invertin, of pepsin and trypsin, of rennet, of forming ammonium carbonate from urea, and probably also ferments capable of rendering cellulose soluble and of breaking up neutral fats into fatty acids and glycerin. The liquefaction of gelatin, which forms one of the distinguishing cultural characteristics of many bacteria, is a process of peptonization by peptic and tryptic ferments, which, indeed, have been isolated, although not in a pure state, from bacterial cells.

The products of putrefaction are known only in part, but even the known products are very numerous. The simplest end-products of putrefaction are carbon dioxide, water, hydrogen, nitrogen, and ammonia, to which may be added nitrites and nitrates and the gases, sulphuretted hydrogen, phosphoretted hydrogen, and marsh gas. But before the end-products are reached a host of intermediary substances are formed. Among these transitional putrefactive products may be enumerated the following: peptones and other proteid substances; nitrogenous alkaloidal bases called ptomaïns or cadaveric alkaloids, as collidin, neuridin, neurin, cholin, cadaverin, putrescin, spermin, saprin, gadinin, peptotoxin, mydatoxin, mydin, methyl-guanidin, trimethylamin and other amines; other nitrogenous substances, as leucin, tyrosin, taurin, glycocoll; methylmercaptan; tryptophan; aromatic substances, as phenol, indol, skatol, hydroquinon; and organic acids, as formic, acetic, butyric, lactic, propionic, palmitic, caproic, succinic. Processes of hydration, dehydration, oxidation, and reduction are concerned in the formation of putrefactive products.

Many different kinds of bacteria take part in producing ordinary putrefaction, some aërobic, others anaërobic, one kind preparing the way for the growth of another. The presence or absence of free oxygen has marked influence upon the course and products of putrefaction. It is especially the anaërobic bacilli which produce the most malodorous substances and gases. The course and products of putrefaction are manifold, and vary with the nature of the material, with the temperature, the access of oxygen, and other external conditions, and especially with the character of the micro-organisms present.

In considering subsequently food as a source of infection and intoxication attention will be given to the poisonous ptomaïns of putrefaction. The epithet aërogenic is applied to bacteria which produce gas.

Among the vital activities of bacteria may be included the production of pigments. The epithet "chromogenic" is applied to these color-producing bacteria. Most of them are harmless. The staphylococcus pyogenes aureus, the bacillus pyocyaneus, and a few others are pathogenic. All of the colors of the spectrum, as well as black, are represented in the different tints which may be formed by chromogenic bacteria. Whether the pigment is in the protoplasm or the enveloping membrane of the bacterial cells or in the surrounding medium cannot always be determined, but that it may be in the medium, and even diffused a wide distance from the bacterial growth, is apparent in the cultures of some chromogenic bacteria. As a rule, only the part of the culture exposed to the air presents the color, but with some species it may be in the deeper parts, and even absent from the exposed surface when present in the depth.

Several interesting species of bacteria capable of producing light, and therefore called photogenic or phosphorescent bacteria, have been obtained from decomposing phosphorescent fish and meat and from sea-water.

The power of producing disease which characterizes the pathogenic bacteria is a manifestation of vital activity which will be most conveniently considered subsequently in connection with infection.

Here will be explained only the meaning of the terms "saprophyte" and "parasite." A parasite is an organism which lives in or on another living organism, called the host. The name "parasite" is often restricted to such organisms as live at the expense of their host, and the name "commensal" or "messmate" is applied to an organism which lives simply as a tenant or coinhabitant, but not at the expense—and it may be even for the benefit—of the host. The latter mode of life is called symbiosis. The term "saprophyte" was originally applied to plants which grow on decaying vegetable and animal matter, but in bacteriology it designates all bacteria which grow outside of a living host. An obligate parasite is an organism which develops only within a living host. An obligate saprophyte is one which grows only outside of a living body. A facultative parasite is one whose ordinary mode of life is saprophytic, but which is capable of a parasitic existence. A facultative saprophyte is one whose ordinary existence is parasitic, but which can grow in dead organic matter. The bacillus of leprosy, the spirillum of relapsing fever, are, so far as their life history is known, obligate parasites; the bacilli of anthrax, tetanus, and malignant œdema are facultative parasites. The great majority of bacteria are obligate saprophytes. We may by special methods be able to cultivate in our artificial media parasitic bacteria, as, for example, the bacillus tuberculosis, which we suppose cannot find, or at least only exceptionally can find, natural conditions suitable for growth outside of the living body ; and such bacteria are generally regarded as obligate parasites. No sharp distinction, therefore, can be made between obligate parasites and facultative saprophytes. For some parasitic bacteria we cannot say whether their natural habitat is inside or outside of the body.

DISTRIBUTION.

Bacteria are almost universally distributed in the upper layers of the ground, in surface waters, in the air, and on objects exposed to the air. They are especially numerous where there is dead organic material. Only a small fraction of these widely-distributed germs are pathogenic. Most of them are harmless and many are useful.

Bacteria cannot ordinarily find suitable conditions, especially the requisite moisture, for multiplication in the air. They are wafted by the air from the ground and objects upon it, but it is a fact of fundamental hygienic importance that the air is incapable of detaching bacteria from moist surfaces except occasionally in spray. Hence only such germs as can withstand complete desiccation are found in a living state floating in the air. Whatever creates dust brings bacteria into the air. The air in the neighborhood of human habitations is particularly rich in bacteria and fungus-spores, whereas sea air at a considerable distance from land is nearly or quite free from micro-organisms, this distance naturally varying with the direction and strength of the wind. The air at high altitudes may be nearly or quite free from germs.

Bacteria do not seem to occur to any extent in the air as single, detached cells, but rather as clumps or attached to particles of dust, so that in a perfectly quiet atmosphere these particles containing bacteria rapidly settle upon underlying objects, and are easily filtered out by passing the air through porous substances, such as cotton-wool, or sand.

The ground, unlike the air, is the resting or the breeding place of a vast number of species of micro-organisms. Instead of a few bacteria and fungi in a litre, as ordinarily with the air, most specimens of soil contain thousands, and often hundreds of thousands, of bacteria in a cubic centimetre. Fränkel found the virgin soil almost as rich in bacteria and fungi as that around human habitations. The principal home of the bacteria of the outer world is in the ground. It is here that they live and flourish and carry on those great processes of putrefaction, fermentation, and nitrification so essential to the economy of nature.

This vast richness in micro-organisms belongs, however, only to the superficial layers of the earth. Where the ground has not been greatly disturbed by human hands there is, as a rule, at about three to five feet below the surface, an abrupt diminution in the number of living organisms, and at the depth where the subsoil water usually lies bacteria and fungi have nearly or entirely disappeared. Of course the number of bacteria and the depth to which they penetrate vary with the character, especially the porosity, of the soil and the way it has been treated.

Among the pathogenic bacteria which have their natural home in the soil, the most widely distributed are the bacillus of malignant œdema and the bacillus of tetanus. In infected localities the bacillus anthracis lives in the soil. Mention has already been made of the presence and function of nitrifying bacilli in the ground.

As regards the presence of bacteria in water, an important distinction is to be made between surface waters and subsoil waters. All surface waters contain bacteria, their number depending upon various circumstances, and especially upon the extent of pollution of the water with animal and vegetable waste. The bacterial flora of water includes a great variety of species. On the other hand, water coming from the deeper layers of the soil is under ordinary circumstances and in most places free from micro-organisms, so that such water forms the safest source of supply for drinking-water, provided care be taken that it is not exposed to contamination. There are certain bacteria, particularly the so-called water bacteria, which can multiply actively even in the purest water, but this is not true of pathogenic bacteria, which require more concentrated nutriment and are crowded out by the water bacteria. Nevertheless, such pathogenic germs as the bacillus of typhoid fever and the spirillum of Asiatic cholera may survive sufficiently long in water to render this medium an important distributor of these germs to human beings.

The so-called self-purification of water is due to various circumstances, among the more important of which are the action of sunlight and oxygen, the action of bacteria, algæ and other organisms, sedimentation and dilution.

As might naturally be expected, micro-organisms are present on the skin and exposed mucous surfaces of the animal body, where they are brought by the air and by contact with external substances. Bacteria are carried in large number into the alimentary canal with the food and drink, and are freely inspired with the air. All sorts of bacteria are brought in this way into contact with the skin and exposed mucous surfaces, but most of them are entirely harmless, and are sooner or later removed. There are, however, certain bacteria which are found so constantly or with such frequency in these situations that we can properly speak of a definite bacterial flora of the skin, of the mouth, of the nose, of the intestine, and of the vagina. Thus, in the epidermis and the glandular appendages of the skin we find regularly the staphylococcus epidermidis albus; in the mouth, leptothrix and spirillar forms which will not grow on our ordinary culture media; in the intestine, the bacillus lactis aërogenes and the bacillus coli communis; and in the normal acid secretion of the vagina, the bacillus vaginalis. Other species are usually mingled with these regular inhabitants, among the most common and important intruders being the pyogenic cocci, and in the mouth also the micrococcus lanceolatus. When the vagina contains pyogenic cocci, its secretion is generally altered in reaction and appearance.

These ordinary surface bacteria cannot in health, with intact skin and mucous membrane, penetrate into the circulation and the internal organs, or, if now and then they do happen to make their way into the circulation, they are in health quickly destroyed. The internal organs and fluids are normally entirely free from micro-organisms.

Agencies Injurious to Bacteria.

Injurious agencies, according to their nature, duration and intensity of action, may affect bacteria in various ways. Thus bacteria may be killed outright or stopped in development, or manifest only slow, tardy, and scanty growth, or lose or suffer modification of biological properties, such as the capacity of producing fermentation or disease. The modifications of biological properties will be considered separately.

As has already been mentioned, spores are much more resistant to injurious agencies than are the vegetative bacterial cells, although there are decided differences in the resisting power of the spores of different bacterial species, as well as of the different bacteria in the vegetative state.

Bacteria may fail to grow or may develop only feebly or with involution forms, or be otherwise modified in character, when their nutritive pabulum is insufficient or too concentrated or of improper reaction or of unsuitable composition. In our artificial cultivations we endeavor to provide for each bacterial species that nutriment which offers the best conditions for its development. Whereas many bacteria will grow with almost any kind of food we offer them, others, such as the nitrifying bacilli and certain pathogenic bacteria, require nutriment of a very particular composition and of definite reaction. Absence of sufficient water is one of the most common conditions which hold in check bacterial growth, and this property is utilized in preserving foods by addition of sugar and of salt, by drying, and by various other processes of concentration.

The cessation of the growth of bacteria in nutritive media may be due to the exhaustion of nutritive supply, but in our culture media it is more frequently the result of alteration in the reaction of the medium and of the accumulation of injurious bacterial products. Bacteria may produce various substances which in sufficient concentration are inimical to their own development or to that of other species; and some of these bacterial products are among the well-known antiseptic substances, such as various acids and phenol. On the other hand, some bacterial products, at least in moderate concentration, are distinctly favorable to the growth and functional manifestations of the bacteria forming them or of other bacteria.

Under natural external conditions bacteria are not usually found in pure culture, but are mixed together, and it is therefore important to know how one species may act upon another. These mutual relations are undoubtedly of a complicated nature, and are only imperfectly understood. The struggle for existence among these lowly organisms must often be keen. We have evidence that one kind of bacterium may by its vigor and rapidity of multiplication simply overgrow and crowd out another; that one kind may produce substances hostile to the development of another; that two or more species may grow just as well or even better side by side; that one species may render the soil more suitable for the simultaneous or subsequent growth of another; that two kinds may grow together, but with modification of the properties of one or both—for example, attenuating or increasing pathogenic power. Such mutual antagonisms and friendships as these must evidently play an important, although at present little understood, rôle in bacterial life.

Bacteria have their natural enemies, not only among their own kind, but also among other low organisms, as well as in the higher plants and animals.

With the exception of the limited, but to us most important, group of parasitic bacteria, these micro-organisms cannot live in the living bodies of plants and animals. The power to resist putrefaction has often entered into definitions of life. The inability of bacteria to develop in plants is usually attributed chiefly to the acid reaction of the vegetable juices and to impenetrable vegetable membranes, but it may well be doubted whether the natural immunity of plants to bacterial growth is not due also, in large measure, directly to the vital properties of the vegetable cells. Certain it is that in animals, aside from certain mechanical defences, which will be described hereafter, the living cells of the body furnish the chief weapons to attack invading bacteria; and this is true whether the field of battle be within the bodies of the cells or in the tissues and juices outside of the cells. These antibacterial properties of the cells and fluids of the living animal bear directly upon questions of infection and immunity, and will therefore be more fully considered subsequently.

In this connection the influence of various physical agents upon bacteria may be considered.

Direct sunlight, independently of the heat which it develops, is a powerful destroyer of many bacteria. This action is most intense when the bacteria are in a moist condition, with free access of oxygen and directly exposed to the sun's rays, as, for example, when the bacteria are suspended in water or in a thin layer. Under these circumstances Buchner found water containing 100,000 colon bacteria to be free from living bacteria within an hour. In a few hours, or even less time, direct sunlight may kill anthrax bacilli and spores, tubercle bacilli, typhoid bacilli, cholera spirilla, and many other species. On the other hand, there are bacteria which are not destroyed, or even injuriously affected, by light, and which seek the light. The most decided injurious effect is produced by rays from the violet end of the spectrum. Electric light and diffuse daylight are also injurious to many bacteria, but in far less degree than direct sunlight. Direct sunlight, accompanied often with desiccation and elevation of temperature, is one of Nature's most potent agents for destroying bacteria.

Electricity appears to damage bacteria chiefly by its electrolytic action.

Most vegetative bacterial cells are killed by exposure in the moist condition for fifteen to twenty minutes to a temperature of 60° C.; not a few are killed within this period by temperatures varying between 52° and 60° C.; but some require higher temperatures (the tubercle bacillus 65° C. for fifteen minutes), and rare species are known which will even multiply at 60° C. and over. The pathogenic spores, so far as known, with the exception of those of symptomatic anthrax (Rauschbrand), are killed in a few minutes by boiling, but there are very resistant spores which will withstand boiling for five or six hours. All spores are destroyed within twenty minutes by steam under pressure at 120° C.

Nearly all vegetative bacteria are killed in an hour and a half by dry heat at a temperature of 120° C., but the destruction of spores within this period by dry heat may require temperatures of 140° to 160° C.

Some species of bacteria are killed in a few days by freezing, but many survive for a long time. Prudden found alternate freezing and thawing more injurious than continuous freezing.

The cholera spirillum is the most susceptible of all known bacteria to drying, being killed in a few hours by complete desiccation, whereas other vegetative bacteria may survive in the dried state for weeks and months, but are eventually destroyed. Dried spores may retain their vitality for years, and some probably for centuries.

Vibration, such as can be produced by shaking, is favorable to the development of some bacteria, and is injurious to others.

Bacteria may be killed by subjection to high atmospheric pressure—the bacillus pyocyaneus, for example, by a pressure of fifty atmospheres of CO_2 in six hours, the anthrax bacilli by twelve atmospheres.

An interesting property of motile organisms was discovered by Pfeffer and called by him chemotaxis. Many motile micro-organisms, including bacteria, are attracted or repelled by definite chemical substances dissolved in water. The name "positive chemotaxis" is given to the phenomena of attraction, and negative chemotaxis to those of repulsion. Thus typhoid bacilli and cholera spirilla moving freely in a fluid medium will be attracted into an open capillary tube containing potato-juice or solution of potassium chloride placed in the fluid. On the other hand, they will flee from many acids, alkalies, and alcohol, and to other substances they may be indifferent. The phenomena of chemotaxis cannot be explained by simple physical laws, but relate to the life and motility of organisms.

For the antiseptic and disinfectant uses of various chemical substances, as well as for further information concerning heat as a disinfectant, the reader is referred to the section on Disinfection in the article on Hygiene in Vol. I.

MODIFICATIONS OF CHARACTERS; ATTENUATION OF VIRULENCE.

In a broad sense bacterial species breed true and transmit the ancestral characters through an indefinite number of generations. But, like other organisms, and apparently in more marked degree than is the case with higher plants and animals, bacteria are susceptible of variation, especially with changed conditions of environment. Also, as with the higher forms of life, some species are tenacious of their special characters, while others are more readily susceptible of variation.

Most of the variations with which we are familiar are temporary, and soon disappear after a return to the normal environment, but some become permanent and heritable even after such return, and thus give origin to new varieties, races, or species.

Permanent variations in function occur more frequently and are more easily produced than permanent variations in form. With our present crude classification of bacteria these functional variations often necessitate the establishment of new varieties which would not be recognized as such if we had a more natural classification.

Most, although not all, of the modifications of character which we can produce are referable to injurious influences, such as prolonged saprophytic cultivation of parasitic bacteria, especially when exposed to light and oxygen, scanty or unfavorable food, high temperatures, prejudicial chemical substances, particularly acids and dilute antiseptics, association with other bacteria or their products, the action of certain animal juices, and passage through the bodies of certain animals.

The most common changes in form under these unfavorable circumstances are those which have already been referred to as involution or degeneration forms, and which are to be regarded as pathological. These disappear under more favorable surroundings.

Some bacteria may present considerable diversity in form, particularly in size, when grown in different media, but there are no well-authenticated instances known in which any material change in form has been permanently established, so as to be long retained with return to the most favorable surroundings. When these most favorable surroundings are to be found in parasitic life, and the bacterium has been deprived of virulence, it may not be possible to restore the usual form ; for instance, the micrococcus lanceolatus, which grows usually in pairs or short chains, may by prolonged cultivation grow in long chains, and then it has usually lost its virulence.

A functional, rather than a morphological, variation is the loss of the power of developing spores which the bacillus anthracis may suffer when cultivated for a long time in repeated generations at 42° C., or when subjected to the action of various injurious chemical substances, as potassium bichromate or carbolic acid. In this way an asporogenic variety of the anthrax bacillus may be produced with permanent loss, even under favorable surroundings, of the capacity to form spores.

The cultural characters of some bacteria have been more or less modified, also the motile capacity of bacteria.

Other interesting functional variations have been produced, such as the loss of the power of forming color, of producing fermentation, or of causing disease. Colorless races of the bacillus prodigiosus, of the bacillus pyocyaneus, and of other chromogenic bacteria have been obtained and perpetuated. The bacteria of certain fermentations have been deprived of fermentative property by subjection to high temperatures, although in other respects apparently uninjured and capable of vigorous multiplication. The power of peptonizing gelatin has been in the case of liquefying bacteria lessened or abolished.

To us the most important functional variations relate to alterations of virulence of pathogenic bacteria. By subjection to various injurious influences a number of pathogenic bacteria have been made to suffer attenuation or entire loss of their pathogenic power, and in some instances permanent non-virulent varieties have in this way been established.

Bacteria have been deprived partly or wholly of their virulence by prolonged cultivation in artificial media; by injurious physical agents, as heat, sunlight, desiccation, high atmospheric pressure; by antiseptic chemical substances, as carbolic acid, potassium bichromate, trichloride of iodine; by the blood-serum of certain animals; by products of certain animal cells, especially those in extracts of the thymus gland; by certain bacterial products, for example, cadaverin and undetermined ones in sterilized cultures; by mixture with other bacteria, as, for instance, the anthrax bacillus with the bacillus pyocyaneus, where also the controlling factor is doubtless the action of chemical products; and by inoculation of certain animals.

The more gradual and the less violent is the operation of these attenuating influences, the more permanent is the resulting modification.

Most bacteria become more or less weakened in pathogenic power by prolonged cultivation in artificial media, especially when exposed to light and oxygen. With some this diminution of virulence may be scarcely perceptible after many months, and even years, but with the micrococcus lanceolatus the virulence may have entirely disappeared within a few days.

By cultivating the anthrax bacillus at 42° C. any degree of attenuation of virulence, down to complete loss, may be produced, according to the duration of exposure to this temperature. In this way permanent attenuated and non-virulent varieties of this bacillus may be developed.

In many cases, if not in all, loss of virulence is to be interpreted as loss of power of producing toxic substances. The loss of this function is often, although not necessarily, associated with enfeeblement of other functions, such as the capacity of vigorous and rapid multiplication and of resistance to various harmful influences. Loss of virulence resulting from prolonged cultivation outside the body is generally associated with more vigorous growth, the saprophytic character becoming more pronounced at the expense of the parasitic.

In contradistinction to attenuation, exaltation of virulence may sometimes be secured. This also has been accomplished in various ways, as by the addition of certain chemical substances, by mixture with other bacteria or their products, and especially by repeated passage through the bodies of relatively insusceptible or of susceptible animals.

We have not much information as to the occurrence of natural or spontaneous variations of bacteria independently of such accidental or designed changes in environment as have been mentioned. Nevertheless, there is no doubt that such variations occur. They can be observed especially well in the case of the apparently spontaneous appearance of colorless varieties of chromogenic bacteria. It has been shown that in one and the same culture of the anthrax bacillus and in that of the micrococcus lanceolatus the bacteria may be possessed of different degrees of virulence. Under certain conditions, by a process of natural selection, one or the other of these natural varieties may gain the upper hand and displace the rest, but this explanation, although it has been advanced, is not generally applicable to the various artificial methods of attenuating pathogenic bacteria. We meet under natural conditions both non-virulent and virulent bacilli diphtheriæ and micrococci lanceolati, and we find the pyogenic cocci notably variable in virulence. Natural variations have been observed also with the cholera spirillum, the typhoid bacillus, and the colon bacillus.

The question of the mutability or the fixity of the varieties and species of bacteria has been much discussed. The facts described in this section evidently bear upon this question, but, with our present knowledge, it is not clear just how much weight should be attached to them in answering the question. Doubtless some writers have exaggerated, and others have underestimated, their significance. The fact that most of the modifications, at least those of a permanent character, relate to an increase or diminution of certain functions, and that many are clearly degenerative, would lead us to attach relatively small importance to them in the differentiation of species if we were to be guided, as, unfortunately, we cannot be at present, by the principles employed in determining species in the higher plants and animals. Even if we assign to these modifications the fullest admissible weight, and if we admit that bacterial species and varieties are more liable than those of higher organisms, there is nothing which would justify us in relinquishing the opinion that bacteria are divisible into definite species and varieties, or in supposing that one well-established species can be transformed into a different, already existing, well-established species, as, for instance, the bacillus subtilis into the bacillus anthracis, or the bacillus tuberculosis into the bacillus of glanders. New species of bacteria have been evolved, doubtless, by the same laws of variation and of natural selection which prevail throughout the animal and vegetable kingdoms. From a medical point of view the occurrence of natural variations in the pathogenic powers of bacteria is clearly of great importance in interpreting the varying characters of epidemic diseases.

MARKS OF DIFFERENTIATION.

For the differentiation of bacteria it is necessary to make as complete a study as possible of all their characters, morphological, biological, and chemical. These characters are of varying degrees of diagnostic importance, but even slight and apparently trivial differences may prove to be of significance. Very few bacteria can be positively identified by their morphological characters alone. To these characters must be added, for purposes of diagnosis, a larger or smaller number of other characters, such as staining reactions, cultural properties, chemical products, and pathogenic power. With increasing knowledge of new bacterial species it often happens that marks which were at first thought to be sufficient for the identification of a species are found not to suffice for its separation from other species. Bacteriological literature contains a large number of named bacteria which have been so imperfectly described that nobody could identify them from the description, and there is no doubt that the same species often figures under different names or that the same name has been assigned to different species. This leads to no little confusion, and shows that it is the duty of every one who brings before the world a new species and names it to describe it in all its characters as fully as possible.

The description of the methods employed in studying bacteria belongs to bacterial technology, which does not come within the scope of the present article. It is proper, however, to enumerate here the differential characters which, so far as possible, should be determined for each species or variety of bacteria.

1. Source. Natural habitat, as ground, water, animal body.

2. Morphology. Everything pertaining to size, form, and development, including involution-forms and other variations of form, grouping, as in pairs, chains, clumps, appearance of zooglœa, presence of capsules, development and germination of spores, arrangement of flagella.

3. Staining properties, especially reaction with Gram's (or Weigert's fibrin) stain, and peculiar or irregular staining.

4. Motility, especial care being taken to examine very fresh cultures and cultures in different media.

5. Temperature at which multiplication occurs, including optimum, maximum, and minimum temperatures.

6. Relations to oxygen, whether aërobic, anaërobic, or facultative anaërobic. Relations to other gases, as hydrogen, carbon dioxide, may also be determined.

7. Reaction of media in which growth occurs, and changes in reaction produced by the growth.

8. Cultural characters, in bouillon, nutrient gelatin, nutrient agar, potato, milk, blood serum. The composition of the culture medium may be modified in various ways for special purposes, as by the addition of litmus, sugar, dyes, and other reagents, and additional media may be employed. The points to be noted are too numerous to specify, but among those of fundamental importance are whether or not gelatin is liquefied, milk is coagulated, color is produced, a surface membrane is formed on bouillon, and the appearance of the growth upon potato. A precise description of the colonies in nutrient gelatin is of especial importance.

9. Resistance, both in the vegetative and spore state, to heat, desiccation, prolonged cultivation, antiseptic substances, and other injurious influences. It

is desirable to fix precisely the thermal death-point. The germicidal action of the blood-serum of different animals may also be tested on the organism.

10. Powers of fermentation, tested with various fermentable substances, particularly with glucose and lactose.

11. Pathogenic powers. There should be stated the modes of inoculation, quantity inoculated, duration of disease and symptoms, distribution of the bacteria, and the lesions produced; also the animals which are susceptible or immune, and variations in virulence.

12. Chemical products, both those produced in cultures and those formed in the bodies of infected animals. Of the metabolic, fermentative or putrefactive products formed in cultures it is often desirable to determine not only the kinds, but also the amounts, of gases and acids produced, and especially the production of nitrites and of indol as tested by the nitroso-indol (cholera-red) reaction. The detection of specific toxic and immunizing products is of valuable assistance in studying pathogenic bacteria.

GENERAL CONSIDERATIONS CONCERNING INFECTION.

Definitions.

The word "infection" has been and is still used in various senses.

1. By some writers it is used, in distinction from contagion, to indicate the transmission of disease without personal contact, as through the air and infected things, not persons. This usage, once common in medical writings and still popular, is dying out among physicians, and is no longer supported by the best medical authorities.

2. According to another usage, infection refers primarily to a diseased condition characterized by certain symptoms and lesions, and only secondarily or not at all to the nature and mode of transmission of the morbific agents causing the disease. The etymology of the word—from *inficere*, to taint, to spoil—seems to justify this meaning, but there enters also into the etymological sense the conception of something introduced from without. The morbific agents capable of producing infection in this sense are heterogeneous and not easily classified, including many, but not all, pathogenic bacteria; some, but not all, pathogenic fungi; some, but not all, pathogenic protozoa; a few parasitic worms, notably trichinæ; some, but not all, toxic bacterial products; some poisons produced by other vegetable cells, as abrin and ricin; and some poisons produced by animal cells, as snake-venom. These poisons, produced by vegetable and animal cells, belong mainly to those called ptomaïns, leucomaïns, and toxic proteids. It will be observed that this second definition of infection is essentially clinical. This use is not common, and is not to be recommended.

3. Modern investigations have shown that the most common and typical infectious diseases, according to any signification of the term infectious, are caused by the invasion and multiplication of pathogenic micro-organisms; and it is now customary, at least among bacteriologists, hygienists, and pathol-

ogists, to limit the term infection to the morbid condition caused by the entrance and growth within the body of pathogenic micro-organisms, and to the act or process by which disease is thus caused. Infection in this sense refers to the nature of the morbific agents causing disease, and implies nothing as to the mode of transmission of these agents. The pathogenic micro-organisms which we now know to be concerned in causing infections belong to the classes bacteria, fungi, and protozoa, but we know nothing about the specific agents of infection in many, and these among the most typical, infectious diseases, such as the exanthematous fevers. Infectious diseases, as thus defined, form a tolerably definite and well-recognized group of affections.[1] An infectious disease may be localized or general.

According to this third definition, which is the one followed in the present article, infections are distinguished from intoxications. Poisoning with bacterial products plays the chief rôle in producing the phenomena of an infectious disease, and it is impossible to draw any sharp dividing line between intoxication and infection ; but it is believed to conduce to precision and clearness to regard as agents of infection only such as are capable of reproduction—that is, such as are living organisms—and not to include among these agents chemical poisons, whether produced by bacteria or other vegetable cells or by animal cells.[2]

The old terms contagion, miasm, and miasmatic contagion no longer answer the purpose of modern medicine, but it is necessary to give the student some idea of the meaning attached to them.

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[1] The not very happy term, at least in its English dress, "invasive diseases" (Invasionskrankheiten) has been employed to designate other parasitic diseases, particularly those caused by entozoa. One is certainly reluctant to exclude trichinosis from the list of infectious diseases, as well as to admit to the list most of the diseases produced in human beings by fungi, such as thrush and the dermatomycoses. This reluctance is due to the fact that we cannot well abandon the conception that infection implies a permeation of the body with micro-organisms or their products, such as we generally observe in bacterial diseases. If, however, we take a broad view of the relation of fungi to diseases of some of the lower animals, and especially to plant diseases, we can hardly fail to include pathogenic fungi among infectious organisms.

[2] The difficulties which arise if we include, as is sometimes done, among infectious diseases simple intoxication with bacterial products without the entrance and multiplication of the causative bacteria within the body, are numerous. The bacteria concerned in the not unimportant group of affections caused by the reception of ptomaïns and some other bacterial products formed outside of the body, as in poisoning from decomposing meat, fish, milk, etc., are mostly strict saprophytes, incapable of parasitic growth in the body of the affected animal. It is only certain poisonous products of bacteria which any one would think of considering as infectious agents, and it would not be easy to define which toxic products should be admitted to the group and which excluded. If we admit as infectious agents toxic products of the bacterial cell, there is no reason why we should exclude similar chemical substances, particularly the toxic albumoses, produced by other vegetable cells and by certain animal cells. Poisoning by snake-venom is clinically a typical infection, and is so regarded by Virchow and others, who adopt a clinical rather than an etiological meaning of the word "infection."

If there were any chance, as there is not, of the word "infection" disappearing from medical nomenclature or from popular use, or if there were any good substitute for this word, it would probably not be worth while to try to adapt this old term to our modern conceptions based upon a more exact knowledge of the causation of disease, and it might be left to the vague and loose meaning which has always attached to it in the minds of most people.

A contagious disease is one capable of communication from one individual to another by means of direct or mediate contact. The introduction of the expression mediate contact in this definition is necessary, but it greatly lessens its precision. In ordinary use the epithet contagious is applied only to those diseases which are readily communicable from individual to individual, so that a person affected with the disease is a source of danger to those in proximity to him. When the infectious agent is eliminated from the diseased body only in such a way, as by the fæces, that it is not likely to be directly received by a second individual, but takes a roundabout way, as through the ground and water, to reach another individual, then the disease is not usually called contagious, although under certain circumstances it may be so. Typhoid fever and Asiatic cholera are examples of such diseases.

It is evident that the term infectious diseases is a more comprehensive one than contagious diseases, and is not strictly comparable with the latter, as the former refers to the nature of the agents causing the disease, and the latter to the manner of transmission of these agents from individual to individual. It is also clear that the limits of what shall be called contagion are indefinite and arbitrary. It is not uncommon to speak of any infectious agent as a contagium. Whether or not an infectious disease is contagious in the ordinary sense depends upon the nature of the infectious agent, and especially upon the manner of its elimination from and reception by the body.

Most, but not all, contagious diseases are infectious. Scabies is a contagious disease, but it is not infectious.

A miasmatic disease is one caused by an ectogenous infectious agent neither directly nor indirectly derived from another case of the same disease. The type of a miasmatic disease in the sense defined is malaria or the malarial diseases. The cause of malaria is a protozoon, which multiplies within the body but, so far as we know, is never eliminated from the body in a living state capable of infecting another individual or the locality. The term miasm is sometimes used in a loose sense to designate infectious agents coming from the water or the ground, especially when believed to be conveyed by the air, or even to designate noxious gaseous emanations and undefined effluvia from the ground.

The expression-miasmatic contagious diseases was introduced in order to explain the often complicated, and even yet not thoroughly understood, modes of spread of Asiatic cholera and typhoid fever, and especially to this group was attached a variety of hypotheses, now for the most part overthrown, and which it is not necessary here to mention. In this group of so-called miasmatic-contagious diseases the infectious agent is eliminated from the diseased body in the living state, but in such a way that it is not likely to be directly received by a second individual, and to produce a second infection it must take ordinarily a path, often circuitous and tortuous and not always easily traced, through various external media. This transit may be influenced, in ways which we now only imperfectly understand, by different meteorological and telluric agencies.

EVIDENCE OF THE CAUSATION OF INFECTIOUS DISEASES BY MICRO-ORGANISMS.

The doctrine that infectious diseases are caused by parasitic organisms is centuries old, and, before the discovery of the specific organisms, the evidence in its favor, based mainly upon *a priori* reasoning and analogy, was presented during the first half of this century with great clearness by J. K. Mitchell in this country and in Europe by Holland, Eisenmann, and, above all, Henle. The property of indefinite multiplication which belongs to infectious agents was the strongest point in this logical argument, as we cannot attribute this property to anything except a living organism. The evidence now, however, for many infectious diseases is no longer aprioristic, but is based upon incontrovertible observations and experiments relating to the causative micro-organisms. We owe to Pasteur as the pioneer and then to Koch and to their followers, the great mass of this positive evidence, which has introduced a new era in the history of medicine.

The complete chain of evidence, essentially as formulated by Koch, to demonstrate that a disease is caused by a specific micro-organism, is as follows:

a. The presence of the organism in all cases of the disease and in such distribution as will explain the lesions.

b. The isolation of the organism in pure culture.

c. The reproduction of the disease by inoculation with the isolated organisms.

When all of these conditions have been fulfilled there can be no doubt that the disease is caused by the organism, and, as one of the conditions is the unfailing presence of the same organism in every case of the disease, is caused solely by this organism.

In addition to the points mentioned, important evidence of the causal relation of a micro-organism to a disease may be afforded by the study of certain chemical substances produced by the organism both in cultures and in the animal body. Many bacteria exert their pathogenic powers not so much by their mere presence as by toxic substances produced by them or contained within them, and many of these toxic substances have specific properties by which they can be identified as the products of definite species of bacteria. Certain amorphous toxic products of bacteria and the immunizing and curative proteids especially render valuable assistance in demonstrating the etiological significance of bacteria. Most of these substances, unfortunately, cannot at present be defined chemically, but they can be recognized by their biological attributes, as will be explained subsequently.

1. For the diseases affecting human beings the three conditions above laid down have been fulfilled in tuberculosis, tetanus, anthrax, glanders, and probably gonorrhœa. Probably acute croupous pneumonia should also be here included, but it is still claimed by some competent authorities that genuine acute croup-

ous or lobar pneumonia may be caused by other organisms besides the micrococcus lanceolatus.

Under the name diphtheria have been hitherto included etiologically distinct affections. Most cases of genuine primary diphtheria are caused by the Klebs-Löffler bacillus of diphtheria. It is probable that the cases caused by this bacillus possess clinical and pathological characters distinguishing them from forms of disease hitherto called diphtheria, but produced by other micro-organisms. If we can thus separate as a distinct disease diphtheria caused by the Klebs-Löffler bacillus from other affections which have been called diphtheria, then the former disease is to be included in the group of those in which the three conditions of Koch have been fulfilled. If, however, the name diphtheria continue to be used to designate etiologically distinct affections, then diphtheria will rank with those diseases which can be produced by more than one kind of organism.

2. There is a large class of infections in human beings in which the three conditions of Koch have been fulfilled with the exception of constancy in the presence of the same organism in all cases of the disease. Here also the evidence is conclusive that a specific micro-organism, found properly distributed in the disease, isolated in pure culture, and when thus isolated capable by inoculation of reproducing the disease, is a cause of the disease, but it is not the sole specific cause. For the most part, the diseases which can thus be referred to more than one kind of micro-organism are the various septic and inflammatory affections, which do not present such sharp and definite differential characters as do those more specific infectious diseases which are caused by only a single species of micro-organism. Some of these diseases present clinical and pathological differences in accordance with their etiological differences, whereas others do not. The class of micro-organisms chiefly concerned in the etiology of these diseases embraces the somewhat heterogeneous group called pyogenic bacteria, of which the pyogenic staphylococci and streptococci are the leading, but not the sole, representatives. To this class of affections belong acute ulcerative endocarditis, meningitis, broncho-pneumonia, erysipelas, pyæmia, septicæmia, osteomyelitis, puerperal fever, other septic and localized inflammatory affections, and, in general, the infections of wounds. Most of these names are simply collective names for different diseases which have already received, and are likely still further to receive, more exact definition as the result of bacteriological studies.

3. The next group of infectious diseases embraces those in which a specific micro-organism is found constantly and exclusively associated with the disease, and has been isolated in pure culture, but the disease has not been reproduced experimentally by inoculation of the isolated organism; in other words, diseases in which the first two, but not the last, of the three conditions of proof above mentioned have been met. To this group of diseases belong typhoid fever, Asiatic cholera, perhaps influenza, rhinoscleroma, actinomycosis,[1] and some of the fungous diseases, which will not be here considered.

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[1] Wolff and Israel have apparently succeeded in producing actinomycosis by inoculation

Typhoid fever and Asiatic cholera are the diseases which chiefly interest us in this connection. So far as we know, lower animals are not susceptible to either of these diseases. The bacillus of typhoid fever and the spirillum of Asiatic cholera manifest in lower animals interesting pathogenic properties which elucidate many points in the pathogenesis of these diseases, but their inoculation in animals has failed to reproduce satisfactorily a disease corresponding in essential features to that observed in human beings; and, in view of the known insusceptibility of lower animals to these as well as to many other diseases of human beings, this result is not surprising. It is true that we do not usually produce in lower animals by experimental inoculation the exact counterpart of the corresponding disease in human beings, nor is it necessary or to be expected that we should do so in order to rest satisfied with our experimental evidence; but the so-called experimental typhoid fever and cholera of guinea-pigs and rabbits are too divergent from the natural diseases in methods of production, in course, symptoms, lesions, and distribution of the bacteria, to be accepted as satisfactory reproductions of these diseases as they occur in man.

Pure cultures of the cholera bacillus have been intentionally swallowed by persons who have previously alkalinized the contents of the stomach with sodium bicarbonate. This experiment has been made in several instances by different individuals. In the majority of cases there resulted diarrhœa of moderate severity, unattended by those marked constitutional symptoms which are attributed to the absorption of the specific poison produced by the cholera bacillus. The cholera bacilli multiplied in the intestinal canal and were found in the fæces. In one case reported by Metchnikoff the ingestion by a healthy young man in Paris of one-third of an agar culture of the cholera bacillus, apparently attenuated in virulence by prolonged cultivation, was followed by a typical attack of Asiatic cholera with all of the classical symptoms—frequent, painless, profuse diarrhœa lasting two days, rice-water stools, repeated vomiting, subnormal temperature, muscular cramps, and anuria on the second day. Reaction began on the third day, and recovery was complete. The diarrhœa set in twelve hours after drinking the culture. During the first two days the intestinal discharges contained an almost pure culture of the cholera bacillus. After the fifth day the cholera bacilli rapidly diminished, but delicate methods revealed a few as late as the seventeenth day. No cholera had existed in Paris for months preceding this experiment. This is the only instance, out of more than a dozen similar experiments, in which a severe attack has followed the intentional ingestion of cholera cultures.

The results of these experiments on human beings are not necessarily out of accord with observations as to the occurrence of cholera under natural conditions of infection, and do not compel us to suppose that the cholera bacilli are incapable of producing the gravest forms of the disease. Out of a large

of animals with pure cultures of actinomyces, so that this disease may perhaps properly be classified with the first group. Further experimental work, however, is needed to thoroughly elucidate this subject.

number of persons exposed to infection with cholera only a portion, usually a minority, acquire the disease. Of the latter only a portion, usually a minority, develop the more severe grades of cholera ; others manifest simple choleraic diarrhœa without marked constitutional disturbance, and some may receive the bacilli without experiencing even diarrhœa or other noteworthy symptoms, although the bacteriological examination of their stools reveals the presence of cholera bacilli. In other words, the same infectious material may cause in different individuals all grades of cholera from simple diarrhœa to the gravest algid cholera, or it may even leave the individual unaffected.

We are not informed at present as to the special conditions of individual predisposition or other conditions which enable us to explain this remarkable diversity in the effects produced by the entrance of cholera bacilli into the intestinal canal of different persons.

Both the typhoid bacillus and the cholera bacillus produce specific poisons and immunizing substances, which, as will be explained subsequently, afford significant corroborative evidence that these organisms are the specific cause of their respective diseases.

The following group of infectious diseases affords an opportunity to consider the weight of evidence to be attached to the constant and exclusive association of a specific micro-organism with a disease.

4. In this fourth group of infectious diseases only the first of the three conditions of complete and stringent proof has been fulfilled. Here belong leprosy,[1] relapsing fever, and the diseases caused by protozoa, the most important of the protozoan diseases now known to occur in man being malaria and amœbic dysentery. No medium has thus far been found on which the bacillus of leprosy or the spirillum Obermeieri can be artificially cultivated, nor have we at present any means of cultivating the malarial protozoa and the amœba dysenteriæ. We have not sufficient evidence as to the syphilis bacillus of Lustgarten to justify ranking syphilis in this group of diseases. Certain affections of lower animals, and possibly some in human beings, caused by other sporozoa may, however, be included in this group, but we shall not consider them here.

The constant and exclusive association of a specific micro-organism with the lesions of a disease admits of no other interpretation in harmony with our existing knowledge than that the organism is the cause of the disease. The only other supposition which deserves a moment's consideration is that the disease is the cause of the presence of the organism. There are instances in which we know that a disease is the cause of the presence of bacteria, but in these cases the same bacteria are found independently of the disease in which they happen to be present ; in fact, these secondary invaders are for the most part widely distributed and common species of bacteria. Moreover, we do not find with these secondary bacterial infections one and the same species constantly present in a given disease. We cannot, therefore, here, as in the class of infectious diseases now under consideration, speak of the constant and exclusive association of one and the same specific micro-organism with a given disease. The possibility that a diseased condition may change some entirely different, perhaps saprophytic, bacterium into the species which is found constantly and exclusively associated with this diseased condition is not supported by any established facts of observation or experiment, and is wholly improbable. Again, it is to be urged that whenever we have found a definite bacterial species constantly and exclusively present in the lesions of a disease, and it has been in our power to isolate in pure culture this bacterium, and to inoculate the isolated organism into an animal susceptible to the disease, it has been demonstrated that such bacterium is the cause, never an effect, of the disease.

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[1] Several investigators claim to have cultivated leprosy bacilli, but in no instance, thus far, has this claim been substantiated to the satisfaction of most bacteriologists.

5. Finally, we have a large number of infectious diseases which have thus far resisted all efforts to discover their specific infectious agents. Here belong yellow fever, typhus fever, dengue, mumps, rabies, Oriental pest, whooping cough, small-pox and other exanthematous fevers, syphilis, and some other infectious diseases in human beings. It will be noted that many of these are the most typically contagious diseases, which it might have been supposed would be the first to unlock their secrets, whereas some of the known bacterial diseases were not previously generally regarded as infectious.

It is, upon the whole, improbable that most of the infectious diseases whose specific micro-organisms have been carefully sought for in vain will be found to be caused by bacteria. They are more probably caused by some other class of micro-organisms, for the demonstration and study of which we have not at present the same satisfactory methods as for bacteria.

We have had in view in this section chiefly the infectious diseases of human beings. There are many diseases confined to lower animals which have been proven to depend upon bacteria, such as chicken cholera, hog cholera, swine erysipelas (Rothlauf), symptomatic anthrax or black leg (Rauschbrand). Here may also be mentioned malignant œdema, which may occur in human beings as well as in lower animals. Texas fever of cattle has been shown by Theobald Smith to be caused by an organism belonging probably to the protozoa.

GENERAL ETIOLOGY OF INFECTIONS.

The mere demonstration of a specific micro-organism as the cause of an infectious disease is not a solution of all of the etiological problems belonging to the disease. There are many other etiological factors of importance, some relating to the infectious micro-organism and others to the individual exposed to infection. Many of these factors are often called secondary or accessory causes.

Number and Virulence of Infecting Bacteria.—The number and degree of virulence of bacteria received into the body may be determining causal factors. In the case of some pathogenic bacteria a single bacterial cell may be capable of infecting a highly susceptible animal, whereas with a less susceptible animal a large number of the same kind of bacteria may be required. With some, and probably most, kinds of pathogenic bacteria the dosage is an important matter, infection occurring sometimes only when many thousand bacteria are inoculated. This dosage, however, is largely a question of individual or racial susceptibility on the one hand and of virulence on the other. Moreover, the kind of infection produced by some bacteria varies with the dose. For instance, the micrococcus lanceolatus may possess such a degree of virulence that a small number of the bacteria inoculated subcutaneously into rabbits will produce no apparent effect, a larger number will cause a local abscess only, and a still larger number will set up fatal general septicæmia.

In our laboratory experiments we are familiar with varying degrees of virulence in the case of a large number of pathogenic bacteria, and, although we have less positive information as to similar variations under natural conditions, we know of many instances of this occurrence—for example, with pyogenic cocci, the bacillus diphtheriæ, bacillus typhi-abdominalis, micrococcus lanceolatus, spirillum choleræ Asiaticæ, bacillus coli communis—and we know that influences capable of affecting virulence, such as sunlight, desiccation, saprophytic growth, association with other bacteria and their products, are operative in nature.

Capacity for Saprophytic Growth.—Micro-organisms which are enabled by external conditions to multiply outside of the body evidently increase thereby the chances of infection by a wider distribution and by their greater number. The diseases usually called endemic, which are spread through infection of a locality, such as malaria, anthrax in animals, amœbic dysentery, typhoid fever, and Asiatic cholera, are caused, at least for the most part, by micro-organisms capable of saprophytic development.

The more important diseases of human beings caused by micro-organisms believed to be obligate parasites are tuberculosis, leprosy, relapsing fever, gonorrhœa, syphilis, rabies, and probably small-pox and other exanthematous fevers. Those caused by organisms whose natural habitat is outside of the animal body (facultative parasites) are malaria, amœbic dysentery, anthrax, tetanus, malignant œdema, actinomycosis, perhaps yellow fever. Those caused by parasites which may multiply outside of the body (facultative saprophytes) are typhoid fever, Asiatic cholera, various affections caused by pyogenic bacteria, and, probably to a less degree, croupous pneumonia and diphtheria. This classification is based partly upon the behavior of the different pathogenic bacteria in our artificial cultures, and partly upon observations concerning the modes of spread of the diseases enumerated. Our knowledge concerning the natural conditions of growth of pathogenic organisms and the sources of infection is not for many infectious diseases sufficiently accurate and extensive to enable us to classify them, except provisionally, under one or another of these heads, and for some we cannot even venture a provisional classification.

Vital Resistance of Pathogenic Bacteria.—The power of causing ectogenous infection is by no means confined to organisms capable of saprophytic growth, upon which, therefore, undue stress should not be laid. Obligate parasites possessing sufficient resistance may live a long time outside of the body and become widely distributed upon external objects. They may even be better adapted for prolonged external existence than some organisms capable of multiplication outside of the body. The tubercle bacillus is an example of such a widely-distributed micro-organism finding only exceptionally conditions suitable for growth outside of a living host.

Of course the bacteria which form spores are the most resistant. The only bacteria infectious for human beings which are positively known to develop spores are the bacilli of tetanus, anthrax, and malignant œdema, all killed by exposure in the moist condition for a few minutes to boiling temperature. It is generally stated that tubercle bacilli form spores, which are, however, less resistant to heat than the preceding. The claim that the bacilli of glanders, typhoid fever, and leprosy also form spores has not been established to the satisfaction of all bacteriologists, and the spores of glanders and typhoid bacilli, if they exist, must be much less resistant to heat than the spores of anthrax, tetanus, and malignant œdema. The tubercle bacillus, the pyogenic cocci, and the typhoid bacillus are among the more resistant bacteria which are not proven to form spores.

The vital resistance of micro-organisms to desiccation is, as has already been explained, an important factor in determining the mode of transmission of an infectious agent, particularly the possibilities of infection through the air. The speedy death of cholera bacilli by drying virtually excludes the conveyance of these germs by the air.

Portals of Entry.—These are the skin, the exposed mucous membranes, as the air-passages and lungs, the alimentary tract, the genital and urinary tracts, and wounds of the skin and mucous surfaces. Intra-uterine infection of the fœtus will be considered later. In our laboratory experiments we make frequent use of methods of inoculation which occur only exceptionally or not at all under natural conditions, such as injection of virus directly into the vessels, into the serous cavities, beneath the skin, and forced inhalation of large numbers of bacteria. In fact, we can rarely imitate experimentally the precise conditions of natural infection.

Some pathogenic organisms are capable of causing infection only when introduced in a definite way—as, for instance, tetanus and malignant œdema bacilli into wounds, cholera spirilla, so far as human beings are concerned, into the alimentary canal—and most have a distinct predilection for particular portals of entrance, as is evident in syphilis, gonorrhæa, diphtheria, pneumonia, tuberculosis, etc.; but some of these, such as the tubercle bacilli, can invade any channel. For a large number of infectious agents, such as those of the exanthematous fevers and relapsing fever, we have no positive information as to how they get into the body, and are left to more or less probable conjecture.

The inference that advanced lesions produced by a specific micro-organism at one or more of the situations above enumerated as portals of entry indicate that the organism must have entered there from without the body, is a common one, but not always warranted.

It is interesting to consider the defences which have been set up at the different exposed parts of the body.

The skin, which is the part most exposed, is protected by a dense epidermis, impenetrable when intact to ordinary bacteria, including most pathogenic forms. Hence we recognize wounds of the integument, which may be so slight as to be difficult or impossible of detection, as a most important predisposing cause of cutaneous infection. There are, however, pathogenic organisms which may settle in the healthy skin and produce disease, but it is improbable that there are any micro-organisms which can pass through the healthy skin and thence invade the body without producing any damage at the point of entrance.

The mucous membranes at the orifices of the body, and some elsewhere, as the buccal, œsophageal, vaginal, which are covered with thick laminated flat epithelium, are hardly less efficiently protected by the nature of their covering than is the skin.

The more delicate mucous membranes covered by cylindrical epithelium are so situated as to be less exposed to insults, but even these apparently more penetrable mucous membranes probably do not permit the passage of many pathogenic bacteria without the occurrence of some damage to their integrity. So far as we can determine, however, some pathogenic organisms—for example, the malarial protozoon—can pass through intact surfaces into the interior of the body. We must admit this for the tubercle bacillus, unless we accept Baumgarten's doctrine of inheritance of the bacillus with an indefinite latent period.

There are certain situations, particularly the tonsils and the lymphatic follicles of the intestine, which seem, by the nature of their covering, especially exposed to the invasion of bacteria; but there is reason to believe that this lymphatic tissue is richly endowed with vital properties hostile to the development of most bacteria.

There are in some situations other mechanical protective arrangements besides the thickness of the covering. Ciliated epithelium drives foreign particles toward the natural outlets of the body. Certain canals communicating with the exterior are only occasionally opened, as the urethra and uterus, and the urethra normally under conditions not permitting the entrance of organisms. The bladder and uterus do not ordinarily contain any bacteria. The tortuous arrangement of the air-passages, especially in the nose, the fact that air is directly inspired only for a relatively short distance into the air-tubes, and the presence of ciliated epithelium are serious obstacles to the penetration of foreign particles into the smaller bronchi and the lungs, so that in these in health bacteria are generally either absent or present only in small numbers, whereas the passages above the larynx contain many organisms.

If we were to rely exclusively upon the results of experiments in the test-tube on the germicidal action of the acid gastric juice, particularly the very acid juice of the dog, we should consider this action a formidable obstacle to the passage of many living bacteria into the intestine. Doubtless this acid fluid is a protective factor not to be overlooked, especially as regards an organism so susceptible to acids as the cholera spirillum ; but when we consider the insusceptiblity of many bacteria to weak acids, the relatively slight acidity of the human gastric juice, the absence often of any acid in the stomach, the withdrawal in large measure from the direct and concentrated action of the gastric juice of bacteria contained in ingested masses of food and large volumes of fluids, and the rapidity with which they may pass through the stomach into the intestine, we can understand how micro-organisms, even very susceptible to acids, may find frequent opportunity to enter the intestine. The normal stomach contains bacteria which find no difficulty in living there.

The secretions of mucous membranes in general afford conditions suitable for development to only a comparatively small number of bacteria, and they are distinctly injurious or even fatal to the life of many bacteria.

We have furthermore to consider that micro-organisms find at the very gates of entrance living cells and fluids which destroy many of them, and even if they pass these gates they are likely to be arrested at the nearest lymphatic glands, which are probably especially adapted to overcome many species of bacteria.

The tendency of modern bacteriological investigations has been to assign much less importance than formerly to the air-passages and lungs as the portals for entrance of infectious agents. Typhoid fever and Asiatic cholera, which were formerly referred to infection by the entrance of germs into the air-passages, are now generally attributed to infection through the alimentary canal. Experiments seem to indicate that only such bacteria as are capable of multiplication in the lungs can produce infection through this organ, but we have no right on the basis of these experiments to draw conclusions as to a similar behavior of other classes of micro-organisms ; for example, the malarial plasmodium and the unknown infectious agents of the acute exanthemata. We must also be guarded in applying to human beings results obtained from forced inhalation or direct injection of large quantities of bacteria—as, for example, tubercle bacilli, pneumonia cocci, anthrax bacilli—into the air-passages and lungs.

Infection through the inspired air, which will be subsequently considered, is not synonymous with infection through the air-passages and lungs, as organisms which enter in other ways may grow or be carried down into the air-passages, and those which have been inhaled may be swallowed.

Channels of Discharge.—The manner of transmission of an infectious disease is influenced, often decisively, by the way in which the infectious micro-organism is discharged from the body.

It was once thought that the living body gets rid of invading micro-organisms almost wholly by discharge through the kidneys, intestine, skin, or other emunctory channels, and the old doctrine of critical discharges and the use of eliminative methods of treatment were based upon a similar conception as to unknown morbific agents. We now know that the living cells and fluids of the body may possess or acquire germicidal and antitoxic powers which are far more powerful weapons of attack or defence against invading bacteria than are furnished by any eliminative functions. Hence we are not surprised to learn that micro-organisms are often disposed of in the interior of the body, and are then not discharged at all as living organisms.

The presence of infecting bacteria in the discharges and their situation depend, in the first place, upon the parts of the body occupied by the bacteria in question. Bacteria which themselves or by their products damage excretory organs or channels find the way opened for their elimination through these diseased parts. It has been stated, on the basis of experiments, that bacteria in the circulation are never discharged from the body through the healthy organs. Most pathogenic bacteria conform to this general law, but there are some which may appear in excretions without any demonstrable lesion of the organ through which they have passed. In general, however, our knowledge of the pathological anatomy of a disease and the distribution of the causal micro-organisms enables us to predict in which excretions we are likely to find these micro-organisms.

Thus the specific micro-organisms are in the sputum in cases of pulmonary tuberculosis, pneumonia, diphtheria, influenza, rabies; in the intestinal discharges, sometimes in the vomit, in typhoid fever, Asiatic cholera, dysentery, intestinal anthrax, and tuberculosis; in the urinary or genital secretions in gonorrhœa, syphilis, genito-urinary tuberculosis, puerperal fever; in the discharges of wounds in tetanus, malignant œdema, traumatic infections; in or on the skin in cutaneous parasitic diseases, lupus, leprosy, erysipelas, typhus fever, small-pox, scarlet fever, measles, rubella. We have no information how the specific organisms are discharged in yellow fever, relapsing fever, dengue, and some other infections. The expired breath is free from micro-organisms, except as they may have been mechanically detached in acts of speaking, coughing, sneezing, or hawking. The breath, therefore, is not the dangerous source of infection which it was once supposed to be. The milk may contain the specific bacteria in certain infections, particularly tuberculosis in cattle and those caused by pyogenic bacteria. A considerable number of bacteria are eliminated through the bile.

It will be observed that the typically contagious diseases, in the ordinary use of this term, are in large part those in which the specific organism is thrown off from the skin, whereas those diseases in which the infectious agent is discharged exclusively or chiefly by the intestine are for manifest reasons not likely to be directly conveyed from person to person, especially as it is chiefly these diseases which are contracted by swallowing the infectious material. In uncleanly and crowded quarters, however, direct transmission of Asiatic cholera and typhoid fever from the stools is not particularly uncommon.

In malaria we have no evidence that the plasmodium is ever discharged from the body in a living state, so that a malarial patient cannot under natural conditions infect either directly or indirectly another person or a locality. Malaria, however, can be artificially conveyed by injecting blood of a patient containing the plasmodium into a healthy person.

Sources and Ways of Infection.—The inferences to be drawn regarding the *infected individual* as an immediate source of infection to others are sufficiently evident from what has already been said concerning the modes of elimination and of reception of pathogenic organisms. The indirect dangers from this source are more conveniently considered in connection with the following external sources of infection, which as regards many diseases represent simply ways of transportation of infectious material from an infected individual to another individual. These media are so numerous and varied that only the more common and important ones can be here specified.

Formerly the *air* was considered the principal medium for the diffusion of epidemic diseases. The patient and his discharges were believed to give off emanations infecting the surrounding air. Even in ectogenous infections the air was regarded as the chief carrier of infection. Contagia transmissible through the air were described as volatile in distinction from fixed contagia.

The tendency now is to restrict within comparatively narrow, perhaps too narrow, limits the possibilities of infection through the air. The principal reasons adduced for assigning to the air a relatively unimportant rôle as a carrier of infection are the following: The demonstration of the frequency and efficacy of other methods of infection, particularly of contact infection; the evidence of absence of danger, or the relatively slight danger, of infection from the air in surgical operations and some infections when every other possible infectious source has been excluded; the observation that in laboratory experiments infected animals rarely infect others through the air; the experimental evidence that the air-passages and lungs do not permit the invasion of many kinds of pathogenic bacteria into the body; the demonstration that living micro-organisms are less abundant in the air than was formerly supposed; the infrequency with which pathogenic germs have been detected in the air; the immense volume of air causing rapid dilution of floating germs, taken in connection with the increasing importance attached to the number of bacteria as a causal factor; the proof that only desiccated bacteria can be conveyed with dust into the air, and that cholera spirilla are killed in a short time by complete drying, and other bacteria may be weakened in vitality and virulence by desiccation, to which often is to be added the injurious influence of sunlight and of oxygen.

These arguments justify the conclusion that the dangers from air-infection were formerly very much exaggerated. It is evident, however, that some of the points mentioned, such as the failure to demonstrate many pathogenic germs in the air, and the application to most human diseases of the mode of spread of diseases inoculated into animals, are of little account, and also that neither singly nor collectively are these points pertinent to all infectious diseases. Most pathogenic bacteria can withstand drying long enough to permit their transportation by the air. Most cholera bacilli are killed within twenty-four hours by drying, but, according to Uffelmann, some may survive as long as this under conditions permitting their conveyance with dust, so that the possibility of the transmission of Asiatic cholera through the air cannot be positively denied, although it must be so exceptional as not to enter into consideration in explanation of epidemics of cholera. Pyogenic bacteria, and more especially the pyogenic cocci, have been repeatedly found in the air, particularly in hospitals. Tubercle bacilli have also, although very exceptionally, been detected in the air of rooms occupied by consumptives.

Certainly nobody supposes that gonorrhœa, syphilis, or rabies can be conveyed by the air, but it would be difficult to mention many other human infectious diseases in which the possibility of such conveyance has been absolutely disproven, although there are several which one would be reluctant to admit to this class. The evidence that the malarial germ is carried by the air seems conclusive. Infectious agents thrown off from the skin on dried epidermal scales, as in the contagious exanthemata, can doubtless be transported by the air, although, as would appear, in sufficient concentration to cause infection only for a short distance, which, however, may be increased by crowding of patients and lack of free ventilation.

It is believed by many that the principal, although not the sole, sources of tuberculous infection are the sputa of individuals affected with pulmonary tuberculosis. The tubercle bacilli are resistant, and may readily be transported by the air from the dust of dried material containing them.

The evidence is irresistible that typhoid fever may be conveyed by the drinking-water and milk; but there is less evidence that it can also be transmitted by the air, although the possibility of this must be admitted.

The possibility of the infectious agents of pneumonia, diphtheria, and especially of influenza, being received through the air is generally conceded.

Even where we admit the possibility of air-infection, however, probably in most diseases other modes of infection are more frequent and serious sources of danger; nevertheless, it would be wrong to overlook the possible dangers from the air, the more so as we are in regard to many infectious diseases in the dark as to the extent of these dangers.

The air may be a carrier of infection to other media, as to water, milk, food, clothing, etc., and to other portals of entrance than the respiratory, as to the skin, wounds, and the alimentary tract. It has already been mentioned that the breath in expiration is not ordinarily a carrier of germs of any kind.

The bacilli of tetanus and of malignant œdema are common inhabitants of the *ground*. These bacilli, especially those of tetanus, are found also with great frequency in the fæces of herbivorous animals, such as horses and cattle, and appear to be more virulent in this situation than in the ground, probably because they can multiply and produce toxic substances better here than in the ground. Various pathogenic staphylococci and streptococci have been repeatedly discovered in the ground. The anthrax bacilli, and perhaps the malarial germ, live in the ground in regions where these diseases are endemic, although the malarial germ has never been detected outside of the human body, and it is possible that it resides in some intermediary host.

The ground receives the discharges of all sorts of infectious diseases. Particularly are to be considered diseases in which the germs are contained in the sputum and intestinal discharges, such as tuberculosis, typhoid fever, and cholera. The tubercle bacilli can scarcely find conditions permitting multiplication in the soil, but it has been proven that they may preserve their vitality at least for many months in this situation. The cholera and the typhoid bacilli may more readily find such conditions for multiplication, but in general the ground with its low temperature, its varying degrees of moisture and dryness, its exposure to sunlight, its insufficient nutriment, and especially its vast number of competing saprophytes, is unfavorable for the growth of these or of most other infectious bacteria. Nevertheless—and this is the important point—these bacilli may survive a considerable time in the ground. Experimental tests have shown that the typhoid bacilli may live for at least three to five months in the soil, and the cholera bacilli, although usually in these experiments dying in a few days, may survive two or three weeks. These experiments as to the survival of typhoid and cholera bacteria in the ground do not reproduce all of the natural conditions with sufficient precision to justify definite deductions as to the possible duration of life of these organisms in or on the ground, and there is evidence from other sources that these bacilli may find conditions for longer life in this situation than that indicated by these experiments.

The principal ways in which pathogenic germs may be conveyed to us from the ground are through dust in the air, by surface drinking-water, by certain articles of food, particularly salads, vegetables, and fruits, by insects, and by direct or mediate contact. Micro-organisms from the ground may be carried by the air or in various other ways to all sorts of objects with which we may come into contact. Street dust of cities has been shown to be very rich in bacteria of all kinds, and among these have been found virulent tubercle bacilli, pyogenic cocci, and tetanus bacilli.

Pettenkofer and his followers attribute to the soil peculiar and indeterminate properties and an exclusive rôle in the spread of typhoid fever and cholera, but we cannot discuss here the hypotheses upon which this doctrine rests.

Water as a carrier of infection comes into question in temperate climates almost exclusively as regards typhoid fever, cholera, dysentery, and diarrhœal affections of indeterminate causation, although its agency cannot be positively excluded in some other infectious diseases. Notwithstanding the lingering opposition of a few hygienists, it may be considered as definitely settled that typhoid fever and cholera may be contracted from contaminated drinking-water.

Bacilli presenting the essential features of the typhoid bacillus have been repeatedly demonstrated in water suspected of causing typhoid fever, although such bacilli have been more frequently missed under these circumstances. Nevertheless, the positive identification as typhoid bacilli of bacteria obtained from external sources is so extremely difficult—according to some, practically impossible—in consequence of the large number and wide distribution of pseudo-typhoid bacilli, that it is doubtful how far we can utilize these recorded observations in support of the origin of typhoid fever from drinking-water.

Cholera bacilli have been found by Koch in the water of a tank in India, and during the epidemics of 1892–93 in Germany in the water of the Elbe at Hamburg, in a well in Altona, and at Nietleben in the Saale and conduit-water supplying the insane asylum where a severe cholera epidemic prevailed, and on the irrigation fields receiving the sewage from the asylum and discharging it again into the Saale at a point near the source of supply of drinking-water. The bacilli were also found by C. Fraenkel and Lubarsch in water during the recent epidemic. The experiences of this epidemic have established upon an incontrovertible basis the long-disputed doctrine of the drinking-water origin of cholera.

Many experiments have been made to determine the duration of life of cholera and typhoid bacilli in water, and with divergent results. Most of these show that in natural waters (unsterilized) the cholera bacilli not only do not multiply, but they die within two or three days, but there are several experiments which show that the cholera bacilli may survive a week. Koch found them still alive after eighteen days in a litre of water taken from a well in Altona and kept at a temperature of 3° to 5° C. The typhoid bacilli may live longer, but according to most experiments these too disappear within a few days up to two weeks from natural waters. Both may live for months in sterilized natural waters.

It is to be remembered that it is very difficult in these experiments to imitate all of the conditions of nature, and it may readily happen that these bacilli may find in some nook at the edge of a stream or well or on some floating objects or in a conduit better conditions for life, and even for multiplication, than would appear from laboratory experiments with water.

Water is rendered a source of infection mainly through contamination with the discharges of infected individuals. Hence any water where the possibility of such contamination exists is to be suspected as a source of supply for drinking purposes or household use. Subsoil water derived from the deep strata of the soil, even when the superficial layers of the latter are crowded with bacteria, is ordinarily practically free from bacteria, especially pathogenic species. If the proper care be taken in procuring it, water which has been thus filtered through the ground, is the safest source of supply. Unfortunately, the common way of procuring this water by dug wells exposes it frequently to most dangerous pollution, so that ordinary well-water in many situations is a particularly dangerous source of infection.

There are various household uses of water, aside from drinking, through which it may convey infectious germs.

Experiments have shown that the susceptibility of animals to infection with the typhoid bacillus may be strikingly increased by injecting certain bacteria, and especially their products. Thus the products of the streptococcus pyogenes, the bacillus coli communis, and the common putrefactive bacillus, the proteus vulgaris, render rabbits and guinea-pigs more susceptible to infection with the typhoid bacillus. These facts, as well as other observations, suggest that certain bacteria and products of decomposition in water, in food, and in the intestine may play an important rôle in predisposing to typhoid fever. Vaughan has found that waters suspected of causing typhoid fever are particularly likely to contain poison-producing bacteria. Contaminated water, therefore, may perhaps predispose those drinking it to typhoid fever independently of the presence in it of the typhoid bacillus.

There are various ways beside that through the agency of water by which the germs of typhoid fever and cholera may be conveyed. There is no occasion for adopting any exclusive theory as to the manner of communication of these diseases, of which cases are not uncommon in which the source of infection cannot be traced.

A careful study of the origin, spread, and distribution of epidemics of cholera or of typhoid fever will often indicate whether or not the epidemic is traceable to infection of the drinking-water. It is especially the explosive outbursts of cholera which are attributable to infection of a general source of water-supply. Here many persons in different parts of the locality supplied by the same water are simultaneously or in quick succession attacked. A second type of cholera epidemics is represented by those in which cases of the disease develop more slowly in scattered groups or foci, especially among the poor living in crowded and insanitary quarters. Here the infection is traceable to cholera patients, less frequently, however, to direct contact with them or their discharges than to infection from contaminated clothing, bedding, furniture, food, insects, etc. These two types of cholera epidemics often become combined or one may merge into the other.

Many bacteria preserve their vitality in *ice* for a long time—the typhoid bacillus, for example, for 103 days in Prudden's experiments. Hence ice should not be procured from water which is unfit for drinking purposes. Uffelmann and Renk have found that cholera bacilli do not survive in ice longer than six or seven days. Cold weather, however, does not prevent outbreaks of cholera, as several severe winter epidemics have occurred when the temperature was below the freezing-point.

Unlike the external sources of infection thus far considered, many articles of *food* afford excellent nutritive media for the growth of a number of species of pathogenic bacteria, and this growth may occur without appreciable change in the appearance or taste of the food. The danger from infection from this source comes into consideration for uncooked or partly cooked food, and for food which, although it may have been thoroughly sterilized by heat, is allowed to stand a considerable time before it is used. Milk, in consequence of its extensive employment in an unsterilized state, its exposure to contamination, and the excellent nutritive conditions which it offers to many pathogenic bacteria, should be emphasized as especially liable to convey certain kinds of infection.

There is strong evidence that the germs of tuberculosis, typhoid fever, and cholera, and probable evidence that those of scarlet fever and diphtheria, may be conveyed by the milk. Tuberculosis is in some regions as prevalent among cows as among human beings, and tubercle bacilli may be in the milk not only when the udder is tuberculous, but when it is healthy, or at least when tuberculous nodules in it are so small as readily to escape detection. There is no evidence, however, that tuberculosis is conveyed by the milk of tuberculous women, although if, as rarely happens, the mammary gland be tuberculous, such an occurrence would not be improbable.

Here may be mentioned the not uncommon instances of poisoning, often of a large number of people (Massenvergiftung), from the ingestion of decomposing or altered fish, mussels, oysters, sausage, canned meats, ham, milk, cheese, and ice-cream. These are due to intoxication, and chiefly to the class of bacterial poisons called ptomaïns produced in the early stages of certain kinds of decomposition. In certain cases of poisoning with milk, cheese, and ice-cream Vaughan has demonstrated a toxic ptomaïn which he calls tyrotoxicon. It is not improbable that the bacteria producing these poisons may continue their activity in the alimentary canal, but there is no evidence that these bacteria, unaccompanied by their toxic products, are harmful when ingested by human beings. It is uncertain whether any of these bacteria are capable of causing genuine infection, although in some fatal cases of these intoxications bacteria have been found in the organs of the body. The best studied of these invading bacteria is the bacillus enteritidis of Gärtner, to which, in fact, he attributes the disease in which it was found.

Insects, especially flies, may assume the sensational rôle of messengers of infection. They may carry infectious agents not only to persons, but also to articles of food and other objects. It has been demonstrated that the bacilli of tuberculosis, typhoid fever, Asiatic cholera, anthrax, and the staphylococcus aureus may pass unchanged through the alimentary canal of flies into the excrement. Sawtschenko found living cholera bacilli in the excrement of flies fed three and four days previously with cholera cultures or with cholera excreta. Mosquitoes may possibly be capable of conveying infection by inoculation.

Various *other objects*, especially such as have been in contact with or proximity to infected persons, as clothing, bedding, furniture, household utensils, animals, the hands, dressings, and instruments of physicians, surgeons, and midwives, and other things too numerous to specify, are manifest and common carriers of infection. Substances which contain contagia are often called *fomites*.

With all sources of infection it is actual personal *contact* with the infected objects which involves the chief dangers.

In this connection a few words may be said concerning infection with *pathogenic bacteria found in healthy persons* more or less frequently, some so constantly that they belong to the normal bacterial flora of the body. The unhappily chosen term "auto-infection" is sometimes given to some of these infections. The bacteria which are most commonly concerned are the micrococcus lanceolatus, pyogenic cocci, especially streptococci, and the bacillus coli communis, but occasionally other pathogenic species, are represented.

Members of this class of bacteria are the ones especially concerned in *secondary* and *mixed infections.* Infectious diseases which cause lesions of mucous membranes, such as tuberculosis, diphtheria, scarlet fever, typhoid fever, open the way to the invasion of these bacteria both by these lesions and by reducing the resistance of the body. The secondary streptococcus infections add to the gravity especially of tuberculosis, diphtheria, and scarlet fever, in which they are very common. In healthy persons these pathogenic bacteria are not likely to do any harm by their presence in the alimentary canal or on other exposed surfaces. These bacteria may, however, under conditions not at present understood, acquire unwonted virulence within the body.

The presence in the mouths of 15 to 20 per cent. of human beings of the micrococcus lanceolatus in a sufficiently virulent state to kill rabbits, and still more frequently in a less virulent condition, bears upon the causation of croupous pneumonia, and doubtless in the sense that predisposing conditions are necessary in many persons to permit the infection of the lungs with this organism.

The occasional existence in the mouth of non-virulent bacilli, apparently otherwise identical with the Klebs-Löffler bacillus of diphtheria, is a significant fact, but not at present clear in its interpretation in relation to the causation of this disease.

Germinal and Intra-uterine Infection.—The embryo may be infected through an infectious agent in or attached to the ovum or in the semen. This is called *germinal* infection. The best-established instance of this is found in an epizootic disease, called pebrine, destructive to silkworms and caused by minute parasitic corpuscles regarded by Balbiani as sporozoa. These peculiar parasites occur both in the ovum and in the semen, and it has been proven by Pasteur and all who have investigated the subject that ova containing them are capable of regular development and generate silkworms infected with the same micro-organisms.

The evidence seems conclusive that congenital syphilis is usually due to germinal infection, and that it may spring from either a syphilitic father or a syphilitic mother.

The possibility that the bacilli of tuberculosis and of leprosy may be transmitted by germinal infection is strongly urged by some of those who consider inheritance of these germs an important factor in causation. There is some evidence that germinal infection of fowls with tubercle bacilli occurs, but no satisfactory evidence of the same mode of infection in mammalian tuberculosis. The bacilli of avian tuberculosis are different from those of human tuberculosis.

In *intra-uterine* or *placental* infection of the fœtus the infectious agents reach the embryo in most, if not all, cases through the blood-circulation by way of the placenta. There are possibilities of infection through the lymph channels of the umbilical cord, through the membranes and amniotic fluid, and through the Fallopian tube and wall of the uterus.

The placenta is a perfect physiological filter without direct communication between the blood of the mother and that of the fœtus. Inanimate particles do not pass through the intact placenta into the fœtal vessels. Pathogenic organisms, however, may be capable of injuring the placental tissue and of growing through unruptured vascular walls.

Both experiments upon animals and observations on human beings have shown conclusively that infectious agents may pass from mother to fœtus through the placenta. The bacilli of chicken cholera and of symptomatic anthrax and pyogenic cocci frequently make this passage, demonstrating that some micro-organisms are better adapted than others to break through the placental barrier. Anthrax bacilli may pass from mother to fœtus, more frequently in some species of animals than in others, but generally in such small number as to require careful search for their detection.

In human beings fœtal infection through the placenta has been observed in small-pox, measles, scarlatina, relapsing fever, syphilis (?), croupous pneumonia, typhoid fever, affections caused by pyogenic cocci, anthrax, and, it is claimed, in one case of Asiatic cholera. In every one of these diseases such transmission, so far as demonstrated, is exceptional, more so in some than in others.

Especial importance attaches to the question of inheritance of tubercle bacilli. Baumgarten advocates vigorously the doctrine that heredity as a causal factor in tuberculosis is mainly inheritance of the bacilli, and not, as is usually believed, inheritance of the soil or predisposition. Baumgarten's doctrine, received at first with great opposition, is gaining adherents, although the weight of authority is still decidedly against it. Instances of congenital tuberculosis, proven beyond doubt, are few; still there are such both in human beings and animals. Johne's first conclusive demonstration of tuberculosis in a fœtal calf was regarded at the time as a unique curiosity. It now appears, however, that similar cases are not very uncommon. Developed tuberculosis of the human fœtus is extremely rare. Baumgarten found in a stillborn infant a tuberculous caseous nodule in the upper cervical vertebræ. More common, but still infrequent, are cases of more or less advanced tuberculosis in infants dying within the first few days after birth. These cases must certainly be regarded as congenital, and probably also the same interpretation must be given to at least some of the cases dying within the first few weeks of extra-uterine life. The frequency of tuberculosis increases very rapidly with each succeeding week after birth, until during the second half of the first year and during the second year of life fatal tuberculosis is very common, and then diminishes in frequency until after puberty.

Birch-Hirschfield detected in a seven months' human fœtus and in the placenta, removed by Cæsarean section from a mother affected with acute miliary tuberculosis, tubercle bacilli both by microscopical examination and by inoculation of guinea-pigs. There were no tuberculous lesions in the fœtus. A similar demonstration has been made by Armanni and by Aviragnet. A larger number of negative results in the same class of cases have been reported.

Until Gärtner's important publication in 1893 comparatively few positive, and many negative, results had been obtained by experiments on animals, both as regards developed fœtal tuberculosis and the presence of tubercle bacilli in the fœtus without tuberculous lesions. Bacilli have been found far more frequently than tubercles in these experimental cases. Gärtner finds that the transmission of tubercle bacilli from mother to fœtus is not at all uncommon in tuberculous mice, canary birds, and rabbits, the last inoculated into the circulation. By inoculating before incubation hen's eggs with bacilli of fowl tuberculosis, Maffucci found that six of eight chicks from the hatched eggs died from tuberculosis in twenty days to four and a half months after birth, precaution being taken to avoid accidental infection. The bacilli remained apparently quiescent in the embryo until birth. These interesting results are not interpreted by Maffucci as due, strictly speaking, to germinal infection, but rather to infection through the area vasculosa, and hence as analogous to placental infection. The possibility of the passage of tubercle bacilli from the mother into the ovum has been demonstrated by Gärtner, who obtained nine eggs from twelve canary birds which had been inoculated in the abdominal cavity with tubercle bacilli. Two of these eggs were proven by inoculation of guinea-pigs to contain tubercle bacilli.

The infrequency of tuberculosis in the human fœtus and the new-born is not a conclusive argument against frequency of placental infection of the fœtus with tubercle bacilli, when one considers the chronic development of the disease, the possible relative insusceptibility of the embryo, the small number of bacilli likely to penetrate the fœtus, and the probability that the conditions are more favorable for placental transmission of tubercle bacilli during the latter part of pregnancy, and especially during parturition, than at an earlier period. We have evidence that tuberculous processes with living and virulent tubercle bacilli may remain latent in the body a long time; and this doctrine of prolonged latent tuberculosis is an essential part of the hypothesis that tuberculosis is often due to bacillar heritage. But notwithstanding the increasing plausibility of the theory of frequent bacillar heritage, many arguments, which cannot be considered here, can be urged in opposition, and the final settlement of the question involves great difficulties not likely to be soon overcome.

We have little information as to the conditions which occasionally permit the passage of micro-organisms from mother to fœtus. These conditions are sometimes to be found in lesions of the placenta, either pre-existing or caused by the micro-organisms themselves or their products. Circulatory disturbances, particularly hæmorrhages, defects in the epithelium of the chorion villi, and areas of necrosis, have been observed with more or less frequency by different observers in cases of fœtal infection, but often no lesion could be detected. During parturition the chances of placental transmission of micro-organisms would seem to be increased, both by circumstances favoring the passage of organisms into the blood of the mother and by increased blood-pressure and ruptures in the placenta.

It is to be noted that, the portal of entry to the fœtus being usually the umbilical vein, micro-organisms would be carried first to the liver and the right side of the heart. Corresponding to this, we find that the organisms are generally most abundant in the liver, and that hepatic lesions are particularly common in congenital infections. Possibly the greater frequency of right-sided endocarditis in congenital heart disease is to be explained in the same way. Pyogenic cocci, which are the bacteria most often associated with endocarditis, break through the placental barrier with comparative ease. Infantile tuberculosis is not, however, nearly so frequent in the liver as in the lymphatic glands, lungs, bones, and joints—a fact which has been urged against congenital transmission of the bacilli. On the other hand, it is especially the frequency of cases of tuberculosis in lymphatic glands, joints, and meninges, without tuberculous lesions at any of the portals of entry, which are considered to support the theory of congenital infection.

With some diseases the fœtus when infected reacts essentially in the same way as the mother, the affection being sometimes more severe, sometimes less so, than in the mother. With some bacteria, however, the characteristic lesions of the disease have not been reproduced in the fœtus, and the conditions appear to be unfavorable for multiplication of the invading organisms. This, however, is not certain, as it may be that the bacilli in such cases have been too short a time in the fœtus to develop and cause lesions. No instance, known to the writer, has been observed in the fœtus of fully-developed anthrax, of croupous pneumonia caused by the micrococcus lanceolatus, or of intestinal lesions by the typhoid bacillus, although in several recorded instances these bacteria have unquestionably invaded the fœtus from the mother. The characteristic lesions have, however, been found so soon after birth as to indicate positively congenital infection. It is interesting to note that the pneumonia cocci and the typhoid bacilli may produce septicæmia without local lesions in the fœtus and congenitally infected new-born. Maffucci found that some pathogenic bacteria inoculated into hen's eggs survived, but did not usually develop until after the birth of the chick. This was true of the bacilli of chicken cholera, to which fowls are very susceptible. This apparent insusceptibility of the embryo to some species of pathogenic bacteria is the more remarkable when we consider that young animals are generally, although not invariably, more susceptible than adults.

Toxic substances, often causing fœtal death, may pass much more readily from mother to fœtus than the micro-organisms producing the poisons.

Predisposition.—A consideration of the general etiology of infections which leaves out of sight important causal factors relating to the individual exposed to infection would be extremely misleading and one-sided. It is convenient to consider these factors under the comprehensive and heavily-burdened term "predisposition." Unfortunately, while conceding the important, often decisive, rôle of individual predisposition for many infectious diseases, we can rarely define the precise nature of the predisposing conditions. We often have to resort to such mere phrases as that the tissues of one person offer better conditions for the growth of a given micro-organism than do those of another.

Instances abound of differences of susceptibility to disease between different species or races of animals, but this racial predisposition is to be distinguished from predisposition as applied to individuals of the same species.

Individual predisposition is not a factor of equal importance for all infectious diseases. It comes into consideration chiefly for those diseases to which the species or race is not in the highest degree susceptible, and for infectious micro-organisms of weakened virulence. There are certain diseases, such as small-pox and measles, to which human beings (unvaccinated) are so susceptible that predisposition becomes a factor of minor importance. There are other diseases, such as tuberculosis and croupous pneumonia, in which predisposition controls the etiology of the disease.

The fact that some individuals are attacked, and others, apparently equally exposed to the danger of infection, escape, is not always due to any especial predisposition on the part of the former. It may be that the germs hit the one and miss the other, and we would have no more right to say that the former are especially predisposed than to say that those who fall in battle are predisposed to bullets and those who escape are bullet-proof. The truth of this is shown by the fact that those who at first escape may be attacked upon subsequent exposure.

The degree of susceptibility to a pathogenic micro-organism influences not only the capacity to acquire the disease, but also the course, severity, and character of the disease. An infectious disease is generally less severe, and, if fatal, kills after longer duration, when there is slight susceptibility. In animals especially, and to some extent in human beings, many species of infectious bacteria remain localized, causing more or less extensive inflammations, in the relatively insusceptible, and the same organisms invade and multiply in the blood, causing general septicæmia, in the highly susceptible. These differences are well exemplified by the differences in the behavior of the micrococcus lanceolatus. Differences in the course and character of tuberculosis may be explained in large measure by differences in susceptibility to the tubercle bacillus; whether also to differences in virulence of the tubercle bacillus is uncertain, but not improbable.

It would appear that varying individual susceptibility plays a more important part in the etiology of most infectious diseases in human beings than it does in animals used for experimentation, although there are many instances of its importance even in the latter. Rats, which are relatively insusceptible to anthrax, have been used to test the influences of predisposing causes of disease.

Individual predisposition may be either local or general; that is, may be due to conditions affecting either a part or the whole of the body.

First in importance among the local predisposing causes are those affecting the portals of entrance of infectious organisms. The situation and the normal defences of these portals have already been considered. There are many obvious ways in which these defences may be weakened or overthrown. Here may be specified wounds, ulcers, inflammations, necroses, hæmorrhages, presence of foreign bodies, alterations in secretions of mucous membranes, trophic disturbances due to impaired nerve-influences. Gastric disturbances, which reduce the acidity and weaken the peristalsis of the stomach, increase the susceptibility to cholera. Many of these defects not only open a passage for micro-organisms, but afford conditions favorable for their development by lessening the vital resistance of the part. A great deal depends upon the invader getting a foothold where it can multiply, if only a little, and produce toxic products which often constitute its real weapons of attack. Besides the grosser lesions mentioned, there may be less evident congenital or acquired abnormalities. Local predisposing conditions are most highly operative when, as often happens, they are associated with general predisposition. Furthermore, the general causes often act by inducing local changes. Finally, when all has been said, we must admit that infectious germs may enter the body, we know not how or where.

After the infectious agents have entered the body they may find local conditions favoring their lodgment and development. Some micro-organisms will not grow in the blood; many show a preference for certain organs and tissues. Often we have no more satisfactory explanation to offer for the various localizations of infectious diseases than to say that some organs and tissues offer more favorable conditions for the life of certain organisms than do others. Sometimes we can explain the localization by the manner of reception of the virus, the vascular relation of the part to infected areas, the size and number of the capillaries, and the readiness with which foreign particles are filtered out.

Injury, inflammation, and other pre-existing disease of a part are important and frequent conditions favoring the lodgment and growth of micro-organisms. For example, bacteria do not readily become attached to the smooth surface of the heart-values, but they may adhere and develop when the valves have been torn or have been roughened by disease. The predisposition of injured joints and bones to the settlement of tubercle bacilli and of pyogenic bacteria is well established, both by clinical and experimental observations. Without some such locus minoris resistentiæ some pathogenic bacteria may enter the circulation and be destroyed without doing any appreciable harm. The anæmic and dry condition of the lung induced by pulmonary stenosis favors the development of pulmonary tuberculosis. The hyperæmic and moist condition associated with mitral regurgitation is comparatively unfavorable to such development, although by no means excluding this disease. The inhalation of foreign particles, as in the case of miners and stone-cutters, predisposes to tubercular disease of the lungs. Bronchial catarrhs, broncho-pneumonias, especially those accompanying measles, whooping cough, and some other diseases, are also local predisposing causes of tuberculosis; but here it is not easy to separate local from general predisposition.

Organs and parts of the body may inherit special vulnerability to certain infections.

Susceptibility to certain infectious diseases may be manifest in races and families. The negro race is less susceptible to yellow fever than the white. Algerian sheep are in large measure insusceptible to anthrax, which is very fatal to other sheep. It would seem, as regards some infectious diseases, as if by a process of natural selection relatively insusceptible races were developed. The frightful mortality of measles freshly introduced among uncivilized peoples, as in the Sandwich and Fiji Islands, is usually cited as a case in point. Black rats are more resistant than gray, and gray rats more resistant than white, to anthrax.

The factors concerned in general predisposition are for the most part less tangible than those of local predisposition. Here also we distinguish congenital and acquired predisposition. Age is a predisposing condition. Certain infectious diseases are most common in infancy, others in adolescence or in maturity or in old age. As a rule, young animals are more susceptible to infections than old ones. There is, however, a special insusceptibility of sucklings during the first months of life to certain infectious diseases, such as scarlet fever, measles, mumps. It is questionable whether there is any difference in predisposition to infection between males and females, except as regards infections directly related to sexual functions.

Impaired vitality and nutrition of the body may predispose to certain infections. More or less plausible predisposing causes operating in this way are bad and insufficient food, overwork, depressing emotions, exposure to extremes of heat or cold, overcrowding, bad air, and, in general, insanitary surroundings. There are experiments on animals showing the effect of food, hunger, overwork, anæmia, and abnormal temperature in increasing susceptibility. Feser, Hankin, and Müller found that rats fed on bread are more susceptible to anthrax than those fed on meat. Canalis and Morpurgo, and Sacchi rendered relatively insusceptible pigeons highly susceptible to anthrax by hunger. Charrin and Roger increased the susceptibility of rats to anthrax and to symptomatic anthrax (Rauschbrand) by making the animals run a treadwheel several hours a day. Anæmia induced by bleeding has been shown to increase the susceptibility of certain animals to various microorganisms, including the anthrax bacillus, the pneumo-bacillus of Friedländer, and the staphylococcus pyogenes aureus. Pasteur made hens, which are normally insusceptible to anthrax, susceptible by artificial refrigeration. Petruschky and Gibier rendered naturally immune frogs susceptible to anthrax by elevating their temperature. Prolonged abstinence from water has been found by Pernice and Alessi to render relatively insusceptible animals more susceptible to anthrax. It is of course to be understood that these various and suggestive results may be explained in different ways, and that they relate only to certain micro-organisms and certain animals, and cannot be directly applied to conditions in human beings.

On the other hand, as regards some diseases—for example, typhoid fever—well-nourished, robust young men are at least as susceptible as those with impaired nutrition.

Diabetes mellitus markedly increases susceptibility to infections, particularly with tubercle bacilli and pyogenic micrococci. By feeding white mice with phloridzin, which produces glycosuria, Leo rendered these animals highly susceptible to glanders, from which they are normally immune. The influence of certain infectious diseases in favoring secondary and mixed infections has already been referred to.

Various chemical substances introduced into the body, such as lactic acid in the case of symptomatic anthrax and tetanus, and particularly certain bacterial products, may increase susceptibility to some infectious organisms. Substances, such as pyridin, which destroy the corpuscles and break up certain albuminous constituents of the blood, may lower the resistance to certain infections. Certain fermentative and putrefactive processes in the stomach and intestine may by auto-intoxication increase susceptibility to infection.

Narcosis may impair resistance to some infections. Klein and Coxwell made frogs and rats highly susceptible to anthrax by narcosis with ether and chloroform, and similar results have been obtained with curare, alcohol, chloral, morphine, and upon other animals and with other diseases.

Climate, altitude, seasons, atmospheric humidity, are believed to influence general predisposition, especially toward tuberculosis and tropical diseases.

By repeated exposure to certain infectious agents resistance seems to be increased. Pathological anatomists are, as a rule, less likely to become infected by post-mortem wounds than are those who rarely make autopsies. Differences in individual susceptibility are well illustrated by the fact that some pathological anatomists are very prone to develop necrophilic tubercles (Leichen-tuberkel), whereas others rarely or never acquire them.

For a while after the subsidence of an epidemic insusceptibility of the survivors, including those who have manifested no symptoms of the disease, is often apparent.

Finally, in many cases we are unable to discover any cause for existing predisposition, and can only say that such susceptible persons by inheritance or by acquirement possess cells and fluids unable to cope with invading micro-organisms.

Many of the predisposing factors which we have considered weaken or abolish this power of the living cells to overcome bacteria. We shall consider under the heading of Immunity more particularly this vital power which distinguishes an immune from a susceptible individual.

TOXIC PRODUCTS OF BACTERIA.

The bacteriological study of infectious diseases has led to the conviction that the phenomena of these diseases are due in great part to poisonous substances formed by the causal micro-organisms. Even before the discovery of the specific micro-organisms purely clinical observation had suggested a similar conclusion in the case of many of these diseases. Our positive information on this subject relates at present chiefly to those diseases caused by bacteria, although we have evidence of the formation of toxic substances in some of the infectious diseases not known to be produced by bacteria.

If we compare the distribution of the bacteria in cases of infectious disease with the character of the symptoms and situation of the lesions, we shall find that we are often unable to explain the latter on any other supposition than that they are produced by absorbed toxic products. The most conclusive proof of this is furnished by those diseases in which the bacteria develop only or chiefly locally, sometimes to a very limited extent, at and around the point of entrance, and nevertheless profound constitutional disturbance, perhaps with marked lesions elsewhere, is produced. The best examples of such toxic diseases are diphtheria and tetanus. Next in order are malignant œdema and symptomatic anthrax in animals. Probably Asiatic cholera belongs also to this toxic group.

At the other extreme are the pre-eminently infectious, in distinction from the toxic, diseases. Here we find the blood and organs swarming with bacteria. Bacteriologists are accustomed to restrict the name septicæmia to diseases characterized by the presence of many bacteria in the blood—a usage not altogether in accordance with the common and traditional sense of this word. We must look to the lower animals for conspicuous examples of these septicæmias in the bacteriological sense, although they are not unknown in human beings. Examples are anthrax, chicken cholera, mouse septicæmia, erysipelas, pneumococcus septicæmia. Even here we have evidence of production of toxic substances, for the bacteria causing these affections develop at first locally, disappearing for a while from the circulating blood if they have been at first directly introduced into it, and often they do not make their appearance in any considerable number in the circulation until a few hours before death and after fever and other constitutional symptoms have set in. Nevertheless, as might naturally be expected, we do not find such concentrated and potent poisons in these bacterial septicæmias as in the more purely toxic affections.

Between the extremes mentioned we have all grades of transition, as, for example, local infections without marked constitutional disturbance, local infections with varying, sometimes much, sometimes little, general disturbance, and infections with more or less wide distribution of the bacteria in different organs, with preference for certain organs according to the disease, but without much or any development of the organisms in the blood. Typhoid fever is an example of the last class, which is often characterized by decided evidences of toxæmia.

Many pathogenic bacteria produce specific poisons not only in the animal body, but, fortunately for purposes of investigation, also in artificial cultures, from which they can be separated and studied as to their chemical and biological properties. Evidence of this is that we can poison animals with sterilized cultures of pathogenic bacteria, and sometimes can reproduce in this way many of the characteristic symptoms and lesions of the disease.

Search was naturally first directed to substances of definite chemical composition, more particularly to crystallizable substances which offer a guarantee of chemical purity. This search was rewarded by the discovery of a large number of crystallizable alkaloidal substances called by Selmi ptomaïns. The larger number of these ptomaïns have been obtained from putrefying substances containing a mixture of undetermined bacteria. Some are poisonous, such as hydrocollidin, mydalein, neurin, cholin, muscarin, gadinin, methylguanidin, peptotoxin, tyrotoxicon, in slight degree cadaverin and putrescin; but many are innocuous. Mention has already been made of this class of bacterial poisons in considering poisoning from decomposing food.

We owe especially to Brieger the study of the ptomaïns formed by pure cultures of pathogenic bacteria. From cultures of the typhoid bacillus he obtained a poisonous ptomaïn called typhotoxin; from cultures of the cholera bacillus, which is an energetic splitter of organic substances, he separated no less than six ptomaïns, some poisonous; in impure tetanus cultures he found four ptomaïns—tetanin, tetanotoxin, spasmotoxin, and an unnamed base. He was unable to discover a ptomaïn in cultures of the staphylococcus aureus or the streptococcus pyogenes, but Leber obtained from the former a crystallizable alkaloid called phlogosin, capable of causing suppuration. This substance has not since been found. Pyocyanin is an alkaloid produced by the bacillus pyocyaneus, found in blue pus, and a pathogenic organism of much interest. Pyocyanin and its derivatives are the coloring principles in the cultures of this organism and in blue pus.

No poisonous ptomaïns have been found in cultures of the anthrax bacillus in ordinary media, but Hoffa obtained a poisonous base from cultures on meat, and Martin has separated a poisonous alkaloid from anthrax cultures containing alkali-albumin and from the bodies of animals and a man dead of anthrax.

The four ptomaïns obtained by Brieger from impure tetanus cultures produced spasms when injected into animals, and this result seemed to render this line of investigation promising.

It was found, however, that the toxic ptomaïns in general do not reproduce in animals the more characteristic symptoms of infectious diseases, and that they are absent from cultures of some highly toxic bacteria—for example, the bacillus diphtheriæ. On this account, and more especially on account of the discovery of another class of substances which have been called toxalbumins or toxic proteids, and of which some do reproduce the characteristic symptoms, medical interest has waned as regards bacterial ptomaïns.

Doubtless the injurious effects of pathogenic bacteria are attributable in part to ptomaïns and other definite chemical substances of a poisonous nature, but these substances cannot claim anything like the importance and interest which attach to certain amorphous toxic products of bacteria which can be only very imperfectly defined chemically at the present time. It is not improbable that some of the poisonous ptomaïns are themselves derived from these so-called toxalbumins. The name "toxins" was given by Brieger to poisonous ptomaïns, but it is now often applied also to the toxic proteids, which may be designated "amorphous toxins" in distinction from the "crystallizable toxins," or poisonous ptomaïns.

We owe to Weir Mitchell and Reichert pioneer work in the study of toxic proteids. They found that snake-venom owes its toxic properties to albuminous constituents. Various toxalbumins, such as abrin, ricin, robin, are produced by the higher plants. The chief impulse to the study of bacterial toxalbumins came from the researches of Roux and Yersin in 1888 on the nature of the poison produced by the diphtheria bacillus, and those of Brieger and Fraenkel in 1890 on the poisons of diphtheria, tetanus, and other infectious diseases.

There is evidence that some of the so-called toxalbumins, notably the poison of tetanus, are not of an albuminous nature, and in fact there is no stringent proof that most of the remarkable substances which have been grouped under the toxalbumins are albumins. These amorphous toxic substances can often be precipitated with albuminous substances contained in cultures or the body fluids. It has been considered an open question as to whether the amorphous toxins thus precipitated are really of an albuminous nature or not. Brieger and Cohn have succeeded in separating the tetanus poison almost entirely from admixture with albuminous material, and as this poison has been more thoroughly studied than any other bacterial poison, and is a most typical member of the group of so-called toxalbumins, we may expect that other members of the group will be separated from albuminous substances. In speaking of these amorphous toxins, therefore, as toxalbumins or toxic proteids, it is to be understood that this designation is only a provisional one in the absence of a better name, and even with our present imperfect knowledge is open to objection. Even if it be assumed that they are really proteids, of course this does not afford an insight into their chemical constitution. Chemically we cannot distinguish the toxic proteids from other known proteids, any more than we can define chemically the difference between living and dead albumin.

It would appear that several kinds of proteids are represented in this toxic group, such as globulins, nucleo-albumins, peptones, and albumoses. Some of the most typical are thought by some writers to be ferments or enzymes, to which in action they bear much resemblance. There is, however, little agreement of opinion as to their chemical position. It is by their biological, rather than their known chemical, properties that we are enabled to recognize and distinguish various toxic amorphous products of bacteria.

A distinction, which, however, is not of a fundamental nature, is made between two classes of amorphous toxic bacterial products: (1) those which readily appear in solution in liquid cultures and in the fluids of the animal body, not being firmly bound by the protoplasm of the living bacterial cells;

(2) those which are incorporated in the substance of the bacterial cells and are set free, especially from degenerated or dead bacteria.

It is more especially the readily soluble amorphous toxins, of which typical representatives are the poisons of tetanus and diphtheria, which in contrast to the so-called bacterio-proteins of the second class have been considered to be metabolic products formed by the action of bacteria directly from the proteid material in cultures or in the animal body. At least some of these toxins may, however, be formed by bacteria growing in culture media free from any trace of proteid material, so that it is probable that they are elaborated within the bodies of the bacteria and are set free in the manner of secretions, as we believe the toxic albumins of snake-venom to be formed in the epithelial cells of the poison-glands.

Buchner, to whom we owe the name "bacterio-protein," obtained from the bodies of a variety of bacteria proteid substances with common properties, being characterized especially by solubility in alkalies, resistance to boiling temperature, attraction of leucocytes, as manifested by leucocytosis and suppuration (positive chemotaxis), and pyrogenic power. Aside from these resistant bacterio-proteins, common to many bacteria, some bacteria—for example, the cholera bacillus—may form unstable amorphous toxins, characterized by specific poisonous properties and intimately incorporated with the bacterial cells.

There is evidence that toxic substances formed by bacteria are sometimes quickly broken up into other, often harmless, substances by the bacteria or their products, and in this case their demonstration may become a matter of great difficulty. These toxic products constitute the chief weapons of attack of many pathogenic bacteria. These weapons are needed much more in the animal body than in cultures. There is evidence that these poisons may be discharged more readily under conditions attending the invasion of the body by bacteria, where they are needed by the invaders, than in cultures where they are not needed. Moreover, we know that some pathogenic bacteria may form toxic products in some culture media, and not at all or but little in other media, and also that some may produce poisons in the animal body and but little in artificial cultures. Hence, we are not to suppose that because we are unable to demonstrate specific poisons in certain culture media, such poisons or different poisons may not be formed in other culture media, and more especially under the divergent and more complicated conditions existing in the living body.

In fixing our attention upon the poisonous chemical products of bacteria we must not lose sight of the fact that these are the results of vital activities. It is only in a very few almost purely toxic affections like tetanus that the actual presence and multiplication of the bacteria in the body are not needed in order that the poison may produce the essential features of the disease. In the case of most infectious diseases we can no more dissociate the actual presence, multiplication, and specific vital activities of the bacteria within the body from the disease than we can substitute any chemical substances for the actual presence and growth of the yeast fungi in the production of alcohol from sugar. We cannot resolve bacteriology into toxicology.

Only those amorphous toxins which belong to the more important diseases of human beings can be considered in this article. The best studied are those of tetanus and diphtheria.

The specific tetanus poison has been demonstrated both in pure cultures of the tetanus bacillus and in the bodies of animals and of human beings dead of tetanus. Inoculated into susceptible animals—and there are very few not susceptible—it produces all of the symptoms of tetanus. The disease produced in our ordinary laboratory experiments by inoculating tetanus cultures is almost entirely an intoxication, not an infection. According to Kitasato, the poison is destroyed in five minutes at 65° C., in twenty minutes at 60° C., in one and a half hours at 55° C.; it stands drying at ordinary temperatures; is not injured by dilution with water or bouillon; is sensitive to acids and alkalies. Kitasato obtained liquid cultures of such virulence that 0.00001 ccm. of the germ-free filtrate, corresponding to 0.00023 mgm. of the dried substance from the filtrate, sufficed to kill a mouse with tetanus. Of course only a part of the dried substance is the real poison. So sensitive is the tetanus poison to chemical reagents that Kitasato was unable to find any means of precipitating the poison without considerable loss of toxicity. He therefore expressed ignorance as to the real nature of the poison.

Brieger and Cohn have, however, succeeded in obtaining the poison in a more concentrated and purified form from veal bouillon cultures of the tetanus bacillus. The poison is first precipitated from the germ-free filtrate of the culture by supersaturation with ammonium sulphate. The precipitate which rises to the surface of the fluid contains the poison. This is collected and dissolved in water. From this solution the albuminous substances are removed by careful addition of basic lead acetate with a trace of ammonia, the poison remaining in solution. Peptones and salts are eliminated by dialysis of the filtrate with running water. The dialyzed liquid is then evaporated in a vacuum at a temperature of 20° to 22° C., by which process volatile substances are removed and the poison is obtained in a concentrated form as yellowish, transparent flakes, readily soluble in water, odorless, with only a small amount of ash and without response to Millon's or the xantho-proteic reactions for albumins. It is free from phosphorus, and contains only an imponderable trace of sulphur, due probably to contamination with some other substance. It is precipitated by ammonium sulphate, but not by sodium chloride, sodium sulphate, magnesium sulphate, boiling, and other characteristic precipitants of albumins. Unlike the diphtheria poison, it is not dragged down by calcium phosphate. Brieger and Cohn conclude that the tetanus poison is not an albuminous substance in the ordinary sense. As to its chemical nature nothing is definitely known. It is sensitive to chemical and physical agents, being injuriously acted upon by acids and alkalies, sulphuretted hydrogen, and high temperatures. Even when kept free from exposure to light, air, and moisture it gradually loses its strength. In the dried state it is not destroyed by absolute alcohol, chloroform, acetone, and anhydrous ether. Temperatures exceeding 70° C. completely destroy the dry poison.

The poison, as thus obtained, although from cultures of less primary virulence than those secured by Kitasato, is of almost incredible potency. As 0.000,000,05 grm. of the purified substance killed a mouse of 15 grm. weight, 0.23 milligram would represent the fatal dose for a human being weighing 70 kilo. When it is considered that the minimal fatal dose of atropine for an adult is 130 milligrams and of strychnine is 30 to 100 milligrams, some conception of the terrible energy of this bacterial weapon can be obtained. It is furthermore to be considered that the substance separated by Brieger and Cohn is far removed from absolute chemical purity.

The diphtheria poison has been demonstrated both in cultures and in animals and human beings dead of diphtheria. It has not been separated from admixture with albuminous substances, and is generally regarded as a toxic albumin, although for reasons already stated the propriety of this designation is open to question. As shown by Ouchinsky, the poison, although in a weakened form, may be produced by the growth of the diphtheria bacillus in media free from proteid constituents. The poison thus formed is believed by Ouchinsky to be of an albuminoid nature, as it was contained in substances responding to the reactions for proteids.

The quantity of the poison formed in cultures depends upon the composition, reaction, temperature and age of the culture and the virulence of the bacillus. The poison is most abundant in cultures four to eight weeks old at body temperature. It is obtained in approximate purity by filtering the culture through a Chamberland filter, partly evaporating the filtrate in vacuo at 30° C., and precipitating with alcohol. It is further purified by repeated solution in water and precipitation with alcohol. The amorphous, dry, white powder thus obtained contains only a part of the poison. No process is known by which it can be obtained in purity. The powder gives the characteristic proteid reactions. The poison is soluble in water, easily precipitated by alcohol and ammonium sulphate, is dragged down with fine precipitates, such as calcium phosphate, is only slightly dialyzable, is destroyed in solution at 58° C., when dry will stand 70° C., and in solution is destroyed by direct sunlight and weakened by exposure to diffuse daylight and oxygen. When inoculated into guinea-pigs and rabbits, it produces all of the characteristic symptoms, including paralysis, and the lesions of diphtheria, with the exception of the false membrane. It is of appalling potency, according to Roux and Yersin four-tenths of a milligram sufficing to kill eight guinea-pigs. When injected in a sufficiently small but fatal dose, the poison produces no apparent disturbance for days, and the death of the animal may occur weeks or months afterward with characteristic symptoms and lesions. This remarkable behavior, so unlike that which we are accustomed to attribute to chemical poisons, raises the question whether the poison may not be reproduced in the body after injection, or even whether the substance injected is itself the poison, and may not be something in the nature of an enzyme which leads to the production of the real poison within the body. These questions, which may be raised also regarding the tetanus poison, cannot be answered with our present knowledge.

It has been shown by Welch and Flexner that the diphtheria poison produces circumscribed areas of necrosis in various internal organs, and that the poison which causes the pseudo-membrane is contained within dead diphtheria bacilli.

There are no bacterial infectious diseases other than diphtheria and tetanus, which have been reproduced in all essential features by inoculating animals with the chemical bacterial products, unless one chooses to consider as such the production of abscesses by dead bacteria or their products. We possess, however, knowledge of some of the bacterial products which are concerned in producing characteristic phenomena of other infectious diseases.

The filtrate of old liquid cultures of tubercle bacilli contains various proteids which are but slightly toxic for healthy persons, but are highly so for tuberculous human beings and animals, producing remarkable reactions and modifications of tuberculous lesions. They belong to the bacterio-proteins which withstand boiling, and are derived from the bodies of the tubercle bacilli. Koch's tuberculin contains these various proteids. Masses of dead tubercle bacilli inoculated subcutaneously in animals cause local abscesses. Prudden and Hodenpyl have shown that suspensions of tubercle bacilli, killed by heat and thoroughly washed, produce when injected into the circulation of rabbits regular giant-celled tubercles, differing from tubercles caused by living bacilli in the absence of caseation, and of course of multiplication of the bacilli. The proteins of tubercle bacilli possess in high degree the property of attracting leucocytes. Prudden suggests that, in addition to these positively chemotactic tuberculo-proteins, living tubercle bacilli produce in the body two other classes of poisons—one which causes caseation of tubercles, and another which causes the constitutional symptoms of tuberculous infection.

Mallein is a toxic protein of the glanders bacillus, bearing much the same relation to glanders that tuberculin does to tuberculosis.

Small quantities of virulent cultures of the cholera bacillus inoculated into the peritoneal cavity of guinea-pigs cause death in a few hours, with symptoms of profound intoxication resembling those of the algid stage of cholera. According to the dose and virulence of the cultures and the susceptibility of the animal, the process may be a pure intoxication with only temporary and slight multiplication of the bacilli, or it may be an infection with marked reproduction of the bacilli.

The germ-free filtrate of fresh cholera cultures in bouillon contains little or no poison; that of cultures a month old contains a considerable quantity, probably extracted from the bodies of dead bacilli. The best-known cholera toxins are those derived from the bacilli. These are highly poisonous, as is shown by the fact that the same profound symptoms of intoxication which follow the introduction of the living bacilli may be produced by the dead bacilli injected in slightly larger dose. These amorphous toxins incorporated with the bodies of the cholera bacilli are less stable substances than Buchner's bacterio-proteins. Cultures subjected to high temperatures, however, are still poisonous. By cultivating the cholera bacilli in a medium free from proteid material, Brieger and Cohn have succeeded in obtaining an amorphous cholera poison devoid of the essential reactions of albuminous substances, with the exception of a slight response to Millon's reagent, which they attribute to admixture with a part of the substance of the bacterial cells.

Ordinary living cultures of the typhoid bacillus in tolerably large doses kill animals chiefly by intoxication—sometimes, however, when sufficiently virulent, by infection. According to Vincent, Sanarelli, and Chantemesse and Widal, the typhoid bacillus by combination with various bacteria, some saprophytic, or their products, may become so exalted in virulence as to produce in animals genuine infection, with extensive multiplication of the bacilli. Sterilized cultures of the typhoid bacillus are poisonous. Toxic proteids, which still retain considerable poisonous power after subjection to 100° C., can be extracted from the typhoid bacilli. A highly poisonous toxalbumin has been obtained by Brieger and Wassermann from the organs in two cases of typhoid fever in man. One-tenth of a gramme killed guinea-pigs in twenty-four to forty-eight hours, with reduction of temperature, somnolence, and general prostration.

G. and F. Klemperer have given the name "pneumotoxin" to the poison produced by the pneumococcus (micrococcus lanceolatus). We have no definite information as to the nature of this substance. Most investigators have had little success in determining the existence of definite toxic substances in cultures of the pneumococcus. As a rule, very large quantities of sterilized cultures or of the germ-free filtrate of virulent cultures of this organism must be injected to obtain any evidence of toxicity. The pneumococcus is extremely variable in virulence, and some cultures are more poisonous than others. There is evidence, however, of the formation of substances of considerable toxicity in animals and in human beings infected with the pneumococcus. The blood in many cases of croupous pneumonia is in a high degree poisonous when injected into rabbits, and this toxic property does not disappear immediately after the crisis.

Various interesting toxic and immunizing proteids are produced by the bacillus pyocyaneus, an organism which sometimes causes general infection as well as local inflammations in human beings.

Cultures of the anthrax bacillus in bouillon are devoid of appreciable toxicity; those in defibrinated blood are somewhat toxic. Martin finds that in culture media containing a proteid, such as alkali albumin, capable of being digested by the bacillus, as well as in the living body, the anthrax bacillus produces poisonous albumoses. Hankin also finds albumoses in anthrax cultures of certain composition and at certain temperatures. Hankin's albumoses possess some immunizing and only slight toxic power.

Soluble toxic products are not abundant in ordinary cultures of the pyogenic cocci. Proteids capable of causing suppuration have been separated from these cultures. Rodet and Courmont have obtained substances, probably proteids, of moderate toxicity from old cultures of the staphylococcus aureus. We have clinical evidence that in some human infections with pyogenic staphylococci, and especially streptococci, toxic substances are formed. In accordance with this, Brieger and Wassermann obtained a toxalbumin from the bloody urine of a patient with facial erysipelas, and Stern found the blood of erysipelas patients toxic. Nissen found markedly toxic the germ-free blood of several patients presenting general symptoms from acute suppurative processes caused by the ordinary pyogenic staphylococci and streptococci. There are, indeed, many recorded instances of decided toxicity of the human blood, secretions, and excretions in various infectious diseases.

Capacity to produce local, especially suppurative, inflammation is a property common to a great many bacteria, and especially to such as are weakened in virulence or are introduced into relatively insusceptible individuals. This capacity depends upon the production by the bacteria of positive chemotactic substances; that is, of substances which attract leucocytes toward them. These chemotactic substances are believed to be, at least in many cases, of a proteid nature. Buchner believes that it is especially the bacterio-proteins discharged from degenerating and dead bacterial cells which attract leucocytes, and that the specific toxic products are negatively chemotactic—that is, repel leucocytes. There is evidence, however, that living and vigorous bacteria, as well as dead bacteria, may attract leucocytes. Nor are all dead bacteria positively chemotactic. Massart believes that living bacteria secrete in varying degree special chemotactic substances, positive or negative according to their concentration, or perhaps distinct positive and negative substances, and that these substances are not identical with the toxic products. As a rule, virulent bacteria attract few leucocytes in a susceptible animal, but attract them in large number in an animal naturally insusceptible or rendered so by vaccination. To this rule there are exceptions.

Bacteria or bacterial products when injected into the circulation cause a speedy diminution in the number of leucocytes. In rapidly fatal infections this diminution is often extreme, and may continue until death. If recovery take place, the diminution is generally followed sooner or later by increase in leucocytes beyond the normal number. In fatal infections of protracted course leucocytosis may or may not occur, there being often notable oscillation in the number of circulating leucocytes. When the bacteria produce local inflammations, there is generally a decided increase in the number of leucocytes in the blood. These experimental facts are in accordance with clinical observations in human beings, and indicate the prognostic value to be attached to leucocytosis, and especially to its absence, in certain infectious diseases, as has been demonstrated in pneumonia.

The phenomena of chemotaxis show that it is not only the distinctively toxic bacterial products which are capable of influencing infection. Under the head of Predisposition mention has been made of what may be called predisposing products of bacteria. These are products which, with or without causing manifest symptoms of intoxication, may render the individual receiving them more susceptible to infection, either to the bacteria producing them or to other bacteria. Ordinary saprophytes may produce such products. On the other hand, the products of some bacteria are antagonistic to infection with other bacteria.

The known facts concerning the biological properties, the causal relations, and the chemical products of bacteria have shed much light upon many pathological questions concerning infectious diseases. There remain, however, a vast number of unsolved problems. We must forego a further consideration of the general pathology of infections, as this would exceed the limits assigned to this article.

IMMUNITY: PROPHYLACTIC AND CURATIVE INOCULATIONS.

Under Predisposition we have considered various conditions which influence susceptibility to infectious diseases. It remains now to consider in a more special way the subject of immunity.

Immunity from an infectious disease may be natural—that is, hereditary—or acquired. There are abundant instances of the natural exemption of certain animal species from diseases to which other, sometimes closely allied, species are susceptible. Field mice, for example, are resistant to mouse septicæmia, which is in the highest degree fatal to house mice, and, on the other hand, field mice are susceptible to glanders, to which house mice are refractory.

Prolonged, often lifelong, immunity is acquired by a single attack of certain infectious diseases, as the eruptive fevers, whooping cough, mumps, typhoid fever, yellow fever, and syphilis. There are other infectious diseases—for instance, diphtheria, pneumonia, and Asiatic cholera—an attack of which confers only temporary immunity. Some diseases, particularly erysipelas, leave behind after a time no increased insusceptibility, and there may follow even diminished resistance to subsequent attacks. There is evidence that some degree of insusceptibility, although it may be temporary and partial, follows an attack of most infectious diseases. The process of recovery from an infectious disease is closely related to the production of immunity.

Until Pasteur's discovery in 1880 of vaccination against chicken cholera, vaccination against small-pox was the only instance known of immunity acquired by this procedure. During the last dozen years means have been found of rendering susceptible animals more or less insusceptible to a large number of bacterial diseases. Thus immunity of varying degree has been experimentally produced in animals from the bacteria of chicken cholera, anthrax, erysipelas, symptomatic anthrax, malignant œdema, hog cholera, typhoid fever, hæmorrhagic septicæmia, vibrionic septicæmia, Asiatic cholera, diphtheria, tetanus, pneumococcus infection, pyocyaneus disease, proteus infection, the bacillus coli communis, and the pyogenic staphylococci and streptococci. To this respectable list should be added rabies, the specific cause of which is unknown.

It is by these important discoveries that the problems of immunity have been partly removed from the domain of pure speculation and have been opened to experimental solution. Independently of their bearing upon the theories of immunity, the modern experimental studies of this subject have disclosed many new facts and points of view so interesting and important that they merit consideration in this work.

The principal means which have been employed to produce artificial immunity are as follows:

1. Inoculation of small, not fatal, quantities of living, virulent cultures of the specific micro-organism causing the disease.

2. Inoculation of the living specific micro-organisms partly or wholly attenuated in virulence. The ways by which virulence may be weakened or annulled have already been described (page 17).

3. Injection of products of the micro-organism causing the disease. Use may be made of products contained in or obtained from sterilized cultures or germ-free filtrates of cultures or of products obtained from the body of an infected animal. These substances may be injected when still toxic, or more frequently after diminution or removal of their toxicity. The principal means of reducing toxic power and increasing vaccinating power are heat and mixture with certain animal juices, particularly thymus extract, or with trichloride of iodine and some other agents injurious to bacterial poisons. The so-called chemical vaccines belong to this class. They have a very wide field of application. In some instances partial immunity has followed the introduction of this class of substances into the stomach.

4. Injection of the blood-serum or other fluids from animals artificially rendered immune from the disease; also the ingestion of the milk of such animals. This method is different in principle from the preceding, and the resulting immunity presents important characteristics which will be explained later. As the blood-serum from immunized animals may possess not only prophylactic but also curative properties, it has received the name of curative or healing serum.

The production of immunity by inoculation of a bacterial species other than that which causes the disease, or by the injection of chemical substances not derived from the specific bacteria of the disease, is of such limited application that it will not be considered here.

Frequently two or more of the principles of immunization described are combined in the method adopted. As a rule, various methods can be successfully employed in producing artificial immunity from a given disease.

Our understanding of the process of immunization was greatly advanced by the discovery that immunity, no less than disease, is produced by the chemical products of bacteria, and may be brought about by these substances, not only when they are elaborated after the introduction of the living micro-organisms in the body, but also when they are formed in cultures or in other animals. Hence the distinction between living vaccines and chemical vaccines is not so fundamental as at first glance might appear.

The bacterial products which produce immunity are generally believed to be proteids, and to belong especially to that class of proteids which are derived from bacterial cells.

There is evidence that toxic proteids can confer immunity, but the fact that immunity can be more readily produced by sterilized cultures or filtrates, deprived by heat or in other ways of poisonous properties, than by the unaltered toxic substances has led to the view that the immunizing bacterial products are either distinct from toxic products or are derived from them. In large doses the vaccinating subtances are generally more or less poisonous.

Immunity does not follow at once upon the introduction of the vaccinating substances into the body. On the other hand, the immediate effect of their introduction is sometimes increased susceptibility. There is a period of more or less pronounced reaction before immunity is established, and often the degree of immunity is proportionate to the intensity of the reaction. This period lasts usually several days, but it may be completed in a few hours or may extend to two or three weeks, varying according to the micro-organism and the character and dose of the vaccine employed. If living vaccine be used, there are generally local inflammation and fever, and there may be a mild attack of the disease. It is not necessary that the bacteria introduced should multiply beyond the neighborhood of the point of inoculation. Fever and loss of weight are the most common symptoms of the reaction following the injection of chemical vaccines, which may or may not cause inflammation at the site of injection.

The immunity which is conferred from a disease by injecting into a susceptible animal the blood-serum or other fluids of another animal which has already been rendered artificially immune from the same disease is contrasted in important particulars with that produced by vaccinating with the specific micro-organism or its products. By the former method we introduce fluids which are already endowed with properties upon which immunity depends, whereas by the latter method these properties must first be developed before immunity is established. In accordance with this difference, we find that the blood-serum from an artificially immunized animal protects at once or within a few hours after its introduction, and does not give rise to any notable reaction. This immediately protective effect of immune serum and the absence of disturbing reaction are qualities which suggested the use of such serum in the treatment of infections. The degree of protection afforded is in direct ratio to the quantity of immune serum introduced and to the degree of immunity possessed by the animal from which the serum was obtained. According to Ehrlich's conception, we simply transfer a part of the immunity possessed by one individual to another individual, and this second individual remains protected only as long as the immunity substances which have been introduced are retained within the body. Hence immunity conferred in this way is called by Ehrlich passive immunity, in distinction from the more stable active immunity produced by vaccination with micro-organisms or their products. It is questionable, however, whether we are justified in taking quite so simple a view of the matter. The demonstration is not conclusive that in producing this so-called passive immunity immune serum or other immune fluids do not bring about some change in the body upon which the final immunity really depends. This change, however, whatever it may be, is certainly not comparable to that which leads to the development of active immunity with its antecedent reaction, often severe and prolonged over several days.

Passive immunity (to retain the use of this convenient but not wholly correct term) is of shorter duration than well-established active immunity. It may, however, continue, with diminishing value, for at least two months. Active experimental immunity is of variable duration. When well established it may in the case of some diseases continue at least several years.

So far as the present evidence extends, it does not appear that any very substantial immunity can be produced by injecting the blood or fluids of naturally immune animals.

It is a general law that a given micro-organism and its products are capable of conferring immunity only from that disease which is caused by this organism. An animal may, however, be vaccinated against two or more pathogenic micro-organisms. The products of the tetanus bacillus, for example, vaccinate only against tetanus, so that an animal made immune from tetanus is not thereby in the least protected from diphtheria or any other infection than tetanus. The tetanus antitoxin is the product of a specific reaction of the animal body induced by products of the tetanus bacillus, and, so far as is known, can be produced only by this bacillus; and the same general statement, with some exceptions, is true of the other specific immunity substances. When, therefore, we find that of two bacteria presenting many characters in common—for example, the typhoid-bacillus and the bacillus coli communis—the products of the one will vaccinate against it, but not against the other, the presumption is that the organisms are not identical. Moreover, when we find in animals or in human beings, after recovery from an infectious disease, specific immunizing substances which were not present before the disease, and which, so far as we know, can be produced only through the activity of a given micro-organism, we feel justified in inferring that that micro-organism has been operative in the causation of the disease. It is manifest that we possess in the detection of such specific immunizing substances a valuable method of demonstrating the causal relation of a micro-organism to a given disease. This method has given positive results in human beings for diphtheria, tetanus, and croupous pneumonia. It has been found also that the blood of persons who have recovered from cholera or typhoid fever possesses decided, often very powerful, properties in protecting animals from the pathogenic effects of the cholera spirillum or the typhoid bacillus respectively. Thus Lazarus found in two persons who had recovered from cholera that $\frac{1}{10}$th of a decimilligram of their blood-serum, and Wassermann in one such case that $\frac{1}{100}$th of a decimilligram of the serum protected guinea-pigs from the effects of subsequent inoculation with a quantity of cholera bacilli surely fatal to unprotected guinea-pigs. The protective influence of the blood progressively increased in Wassermann's case for seven weeks after recovery from cholera. Similar protective properties of human blood against the typhoid bacillus have been found after recovery from typhoid fever. These observations are very significant, but their force is somewhat lessened by the fact that the blood of persons who have never had cholera or typhoid fever sometimes possesses similar protective properties, although less constantly and generally in less degree than in the case of those who have recovered a short time previously from these diseases.

A principle of great importance is that immunity may be augmented in degree by the gradual employment of vaccines of increasing strength, followed by the introduction of the virulent micro-organisms or their products, until finally sometimes doses vastly larger than suffice to kill unvaccinated animals can be inoculated. These very high degrees of insusceptibility, however, have been attained only in the case of those toxic diseases, such as diphtheria and tetanus, the immunity from which depends upon an antidotal power of the blood and fluids against the specific poison of the disease.

When we come to test experimental immunity from various micro-organisms by injecting into the protected animal these organisms or their poisonous products, we find that there are two principal kinds of insusceptibility. With one kind the animal is protected against a fatal dose of the specific bacteria without possessing any decidedly increased tolerance of the toxic products of the bacteria. According to some observations, immunity of this kind is in some instances associated even with diminished tolerance of the poisons. This immunity must depend upon restraint of growth or destruction of the bacteria or upon deprivation of their power of producing toxic substances.

With the other kind of immunity the protected animal is insusceptible to the poisonous products of the specific organism, so that even if the organism should survive for a time in the body, it is reduced to the level of an ordinary saprophyte. It has been proposed to limit the term " immunity " to the first kind, and to describe as "poison-proof," but not immune, those individuals who are simply insusceptible to the poison. While recognizing the importance of the distinctions mentioned, it does not seem wise at present thus to limit the signification of the word immunity. Our knowledge of the conditions underlying immunity is not sufficient to determine how fundamental these distinctions really are.

A moderate degree of insusceptibility to the specific poisons of bacteria may be acquired as the result of a simple tolerance of these poisons, but when this insusceptibility exists in a high degree after immunization it is due to the power of the blood and other fluids of the immune body to antagonize these poisons. If the specific poison be mixed outside of the body in proper proportion with the immune blood-serum, it is found that this mixture can be injected into a susceptible animal without manifesting poisonous properties. It has been generally inferred from this observation that the fluids of the immunized animal actually destroy or neutralize the poison. Such fluids are therefore said to be toxicidal, in distinction from bactericidal, and the antidote to the poison is called antitoxin. It has not, however, been proven that the antitoxin acts by destroying the toxin. It may be that both act in an antagonistic manner upon the living cells of the body, the antitoxin rendering the cells insusceptible to the toxin. Experiments of Buchner indicate the possibility of this latter explanation.

We have no definite knowledge of the chemical nature or the source of these antitoxins. They are commonly believed to be proteids of some form. They are less susceptible to the action of heat and other injurious agents than are the so-called toxalbumins. The albuminous precipitates thrown down from antitoxic fluids by alcohol, and more especially those obtained by adding ammonium sulphate or magnesium sulphate, retain in the dried state a considerable part of the antitoxin. In various ways antitoxins may be obtained in a more concentrated form than in the original fluids.

Our principal knowledge of the practically and scientifically important subject of antitoxic immunity relates to tetanus and diphtheria, and to the vegetable toxalbumins, abrin, ricin, and robin, studied by Ehrlich. We owe to Behring and Kitasato and to Ehrlich the pioneer investigations in this line. The primary immunization of susceptible animals from tetanus or diphtheria may be accomplished in various ways by the bacilli or their products partly deprived of virulence by heat, trichloride of iodine, or other agents.

A most important characteristic of artificial immunity from toxins is that by successive injections of increasing amounts of the poisonous substances into the vaccinated animals the antitoxic or immunizing power of the fluids can be augmented to an astounding degree. It is in this way that the immunizing power of the fluids may become sufficiently high to exert curative effects when injected after the introduction into a susceptible animal of the specific micro-organism or its poison. Each fresh injection of the specific poison into the animal undergoing immunization uses up a certain amount of antitoxin, but this primary reduction is followed each time by a rise in antitoxic power. A partial decline follows this elevation, but the curve remains at a level higher than that existing before the injection. This reaction before the permanent level is reached occupies several days. Considerable time, therefore, is required to obtain blood of very high immunizing power. To what height this may reach we cannot at present tell. Behring in 1892 estimated, according to his method, the immunizing value of the blood-serum of a horse which he had during two years rendered increasingly immune from tetanus as 1 to 10,000,000, by which is meant that $\frac{1}{10000000}$th ccm. of serum will protect 1 gram-weight of mouse from the subsequent injection of the smallest, surely fatal, dose of the tetanus poison, or 1 ccm. of serum suffices to immunize 500,000 mice, weighing each 20 grams, or 200 sheep, weighing each 50 kilo.

Artificial immunity from rabies probably depends upon this so-called antitoxic power of the blood and fluids of protected animals, and, as is the case with acquired immunity from tetanus and diphtheria, this power may be increased by successive inoculations with the virus of rabies.

To what extent acquired immunity from other infectious diseases affecting human beings depends upon an antitoxic power of the blood cannot at present be stated. It has been claimed by the Klemperers that this is the explanation of immunity from the micrococcus lanceolatus, which causes croupous pneumonia. Their observations on this point are not in accord with those of several other experimenters. The experimental immunity which can readily be produced in animals from the bacteria of cholera and of typhoid fever has also been referred to the development of an antitoxic power of the blood, but recent investigations indicate that animals protected by vaccination against these bacteria are not insusceptible in any marked degree to their poisons.

The blood of animals protected by vaccination from the pneumococcus, the cholera spirillum, or the typhoid bacillus is capable of protecting susceptible animals from subsequent inoculation with these micro-organisms. This protective power of the blood reaches the highest degree in cases of antitoxic immunity, but it accompanies also artificial immunity, which is unattended by insusceptibility to the specific poisons of the causal micro-organism. It seems to belong to any solid acquired immunity.

There are many infectious diseases acquired immunity from which cannot be explained by the existence of an antitoxic power of the blood analogous to that present in artificial immunity from diphtheria and tetanus. Immunity from anthrax and other septicæmic diseases, accompanied by great invasion of the blood with bacteria, cannot be explained in this way. The antitoxic principle does not furnish an explanation even for natural, hereditary immunity from tetanus and diphtheria. The blood of the hen, which is naturally immune from tetanus, is not an antidote to the tetanus poison, but it may acquire this antitoxic power after injection of this poison, notwithstanding the fact that the hen is not susceptible to it. This is an illustration of the fact that tolerance by the body-cells of specific bacterial poisons may exist without any antidotal power of the blood and fluids against these poisons. It also shows the wide difference there may be between the basis of natural immunity and that of acquired immunity from one and the same disease. The same general statements are true as regards rats and diphtheria.

Difficulty has been found in conferring immunity from diseases caused by the multiplication of bacteria in the intestinal canal. Guinea-pigs and rabbits vaccinated against the cholera bacillus are protected against several times the fatal dose of cholera cultures introduced into the peritoneal cavity, but it is not satisfactorily established that they are protected from infection with cultures introduced by way of the stomach and intestine under the conditions in which this mode of introduction causes infection. Infections and intoxications of animals with cholera or typhoid bacilli inoculated into the peritoneal cavity are so different from the natural diseases in human beings that we must be very guarded in applying to the latter these experimental results.

The discovery by Ehrlich that immunity may be afforded by the ingestion as well as the injection of the milk of immunized animals is an important contribution to our knowledge of immunity and to our resources in the way of prophylaxis and treatment. It bears upon the question of inheritance of acquired immunity.

Acquired immunity may be transmitted from the mother to her offspring.

The question of germinal transmission of acquired immunity not only is of medical interest, but bears also upon the great problem of inheritance of acquired traits. Only paternal inheritance can give a conclusive answer to this question, at least so far as mammals are concerned. In the case of inheritance of acquired immunity from the mother, the possibility of transmission through the placenta rather than through the ovum cannot be excluded.

The only instance known to the writer in human beings in which it has been claimed that artificial immunity may be acquired by germinal inheritance is that embodied in Profeta's law, which asserts that healthy children born of syphilitic parents are protected against syphilis. The evidence, however, is certainly inconclusive that the healthy child of a syphilitic father and a non-syphilitic mother enjoys any increased insusceptibility to syphilis. The experimental data bearing upon this question are too meagre to furnish a definite answer. Ehrlich found that mice born of normal mothers impregnated by males rendered highly immune from the toxic phytalbumoses, abrin and ricin, possessed no immunity whatever from these substances, and he therefore concludes that "the idioplasm of the sperm is not capable of transmitting immunity." On the other hand, Tizzoni and Centanni assert that acquired immunity from rabies may be transmitted, although much lessened in degree, from the immunized father to the offspring of mothers unprotected against this disease, and Tizzoni and Cattani claim to possess the same evidence for tetanus. They interpret these results as in support of the doctrine of inheritance of acquired traits. Similar experiments by other investigators have afforded negative results.

Passing to placental inheritance, we have to consider three possibilities: (1) protection afforded by contracting the disease in utero; (2) protection resulting from the reception of vaccinating substances from the mother (active immunity); (3) protection conferred by the reception of developed immunity substances from the mother (passive immunity). There is no doubt as to the occurrence of the first and third modes of protection. We have already considered sufficiently intra-uterine fœtal infection, which does not bear upon the question of inheritance of acquired immunity. The occurrence of immunity as the result of reception by the fœtus of immunity substances contained in the mother has been demonstrated by Ehrlich for immunity from abrin, ricin, and robin, and by Tizzoni and others for tetanus and rabies. This passive immunity is reinforced after birth by ingestion of the mother's milk, which possesses the same protective properties as the blood. By this reinforcement it continues longer than would otherwise be the case. The observations concerning inherited artificial immunity in human beings from small-pox, syphilis, and, doubtfully, vaccinia, and in animals from sheep-pox, anthrax, symptomatic anthrax, pneumococcus infection, infection with the typhoid bacillus, and some other diseases, have not generally been made under conditions which permit a positive decision as to whether the immunity is the result of vaccination of the fœtus coincidently with that of the mother, or of the simple transmission of developed immunity from mother to fœtus, subsequently reinforced by the mother's milk. When the mother's immunity is induced during pregnancy by recovery from infection or by vaccination with bacterial products, it does not necessarily follow that acquired fœtal immunity is due also to vaccination. It may be so-called passive immunity. The duration of the inherited immunity may afford an answer to this question.

The only instance known in which immunity appears to be received by the mother from the fœtus is that sometimes observed in the healthy mothers of syphilitic infants (Colles' law).

We possess no general explanation of the basis of immunity. It is certain that the various kinds of immunity—the immunity of plants and of animals, the immunity of warm-blooded and of cold-blooded animals, the immunity from ordinary obligate saprophytes and that from pathogenic organisms, immunity from bacterial poisons and that from the bacterial cells alone, natural immunity and acquired immunity—do not all depend upon the same causes. We can give space here to a brief consideration only of those theories of immunity which rest upon some experimental basis of facts.

The leading theories of immunity which more especially concern us may be brought into two classes—one which attributes immunity to the direct and active intervention of the living cells of the body, and the other, which explains immunity by the properties of the extracellular fluids of the body. In the last analysis these properties of the body humors must depend upon the activities of cells, so that we must have recourse either directly or indirectly to cellular functions in any adequate explanation of immunity.

The chief representative of the first class of theories is the phagocytic theory, which has been elaborated by Metchnikoff with a wealth of detail and a clearness and charm of presentation which make this theory the most fascinating and satisfying to the mind of all. It has also the advantage of a wider applicability, if accepted, than any other explanation of immunity.

The phagocytic theory supposes that immunity depends upon the seizure of invading micro-organisms by amœboid cells of the body, and the subsequent destruction of these organisms by digestion or other chemical agencies in the interior of the cells. The polynuclear leucocytes are the most numerous and important phagocytes, but other mesodermic cells may perform the same function. According to this view, the phagocytes are charged with the defence of the body, and engage in a veritable conflict with the parasites. Immunity is acquired when the phagocytes have gained tolerance of the poisons of micro-organisms and are no longer repelled by them.

The main support of the phagocytic theory is found in the difference in behavior toward invading micro-organisms between the leucocytes of a susceptible and those of an insusceptible individual. If the individual is insusceptible, phagocytes, chiefly leucocytes, accumulate around the micro-organisms and take them up, whereas in a susceptible individual this phagocytosis is generally absent or present only in small degree.

Undoubtedly there exists as a general law, to which there are not a few exceptions, this parallelism between phagocytosis and immunity. But in order to prove that insusceptibility is to be explained solely or chiefly by phagocytosis, it must be shown that the micro-organisms have not first been injured by extracellular agencies before their reception by phagocytes, and also that, even if living and virulent micro-organisms are seized and destroyed within cells, they may not also be weakened or killed outside of the cells.

In the opinion of the writer these objections have not been overthrown by the supporters of the phagocytic hypothesis. It is true that Metchnikoff has proven that phagocytes may take up living and apparently virulent bacteria, and that these bacteria undergo degeneration and death in the interior of the cells. But he has not shown that, as a rule, bacteria when taken up by cells have suffered no injury from extracellular agencies; and, inasmuch as we know that fluids of the body may damage bacteria, the phagocytic theory must bring evidence of the exclusion of these agencies. Direct observations which show that bacteria which have been introduced into the body may degenerate and die not only within cells, but also outside of them in the humors and tissues, are many and come from good investigators, and, although vigorously contested by Metchnikoff, must be accepted.

The principles of chemotaxis have been urged both for and against the phagocytic theory. Those who hold with Buchner that it is chiefly or exclusively the proteids derived from degenerating and dead bacteria which attract leucocytes, whereas the toxic proteids repel them, consider that this is the strongest argument against the phagocytic theory. But, as has already been mentioned, there is evidence that also living and vigorous bacteria may secrete positively chemotactic substances, so that we are not inclined to give decisive weight to this line of argument.

It has been urged against the phagocytic theory that in chronic infections like tuberculosis and leprosy, and in some acute infections, notably mouse septicæmia, living bacteria multiply and flourish in large numbers inside of leucocytes and other cells, and often destroy these cells. The supporters of the theory reply that it is not enough that parasites should be simply received by leucocytes: the intracellular agents of destruction must be operative. The phagocytes have done their best, but these bacteria adapt themselves to the medium within the cells and triumph over them.

We cannot therefore assign to phagocytosis the exclusive rôle which is given to it by Metchnikoff in the explanation of immunity. That it plays a part cannot be doubted; the only question is, How important a part? To regard the phagocytes simply as scavengers and crematories for dead micro-organisms is not in accordance with the undoubted fact that they may seize and destroy living bacteria. Some think that the phagocytes complete the work of destruction already begun outside of them; others believe that micro-organisms perish equally inside and outside of the cells, the conditions being perhaps more unfavorable for them within the cells; and others hold that the antibacterial properties of the extracellular fluids are produced or increased through the agency of leucocytes and other cells. Kossell believes that nucleinic acid contained in or derived from leucocytes and other cells is the chief bactericidal agent. It has been held that leucocytes may form a wall around micro-organisms and mechanically hem them in.

There are many theories which attempt to explain immunity by the properties of the body humors.

It has been urged that immunity may depend upon a lack of nutritive material capable of assimilation by the micro-organism in question, which therefore dies of innutrition. This explanation may apply to some instances of natural immunity—for example, from ordinary saprophytes—but experiments have made it impossible to accept it as the explanation of most cases of immunity, especially of acquired immunity.

The process of immunization and the phenomena of immunity already described necessitate the rejection of the retention hypothesis, at least in its original form, according to which acquired immunity was thought to depend upon the retention for an indefinite period within the body of products of the micro-organism from which protection has been acquired.

The power of the fluids, particularly the blood, of destroying or antagonizing the action of the specific poisons of micro-organisms is the explanation of acquired immunity from a certain number of toxic diseases already considered. Indeed, this antitoxic power is the most thoroughly demonstrated explanation of immunity which we have, but it is applicable only to a limited number of diseases—exactly how large a number we cannot at present say—and apparently only to acquired immunity.

Of the humoral theories of immunity, the one which deserves most consideration for diseases in which the antitoxic power of the blood does not come into question is that which attributes immunity to a direct germicidal action of the blood and fluids of the body. Since the important investigations of Nuttall upon the power of the blood and other fluids to kill bacteria in a short time, this theory of immunity has had many supporters.

Buchner has given the name of "alexins," and Hankin that of "defensive proteids" to the bactericidal and antitoxic substances of the blood and body fluids. Bactericidal alexins are believed to be proteids of an unstable nature. They lose their characteristic property when deprived by dialysis of their combination with certain salts. Vaughan has shown that solutions of nuclein are powerfully germicidal. According to Kossell and to Vaughan, the most important bactericidal agent in the body is to be found in nucleinic acid or in nucleins. The bactericidal power of blood belongs to the serum independently of the presence of cells, and is tested by mixing with the sterile blood-serum or other fluids outside of the body a known number of bacteria, and determining quantitatively, by well-established methods at definite intervals of time, whether the bacteria have multiplied or been arrested in development or have died. It is found that the blood-serum varies in bactericidal power according to the animal from which it is derived, and according to the species of bacteria with which it is tested. According to these varying conditions there are all grades of action, from entire absence of bactericidal power to a capacity of killing many hundred thousand bacteria within a few hours.

The chief difficulty with this theory of immunity has been to establish any such correspondence between insusceptibility and bactericidal power of the blood and fluids as exists between phagocytosis and immunity. We find, on the one hand, that the blood-serum of some animals susceptible to a given bacterium is destructive in marked degree to this organism, and, on the other hand, that the serum of some animals insusceptible to a bacterial species is devoid of notable bactericidal power over this species. There are many cases in which we cannot at present explain immunity by any demonstrated antibacterial action of the blood and fluids, but this is not saying that such action is absent in these cases.

There are, however, several striking instances of correspondence between immunity and bactericidal power of the blood. This is true in some cases of natural immunity, but more convincing are those cases in which the bactericidal capacity is developed as an accompaniment of acquired immunity. The blood-serum of normal guinea-pigs possesses relatively little bactericidal power over the vibrio Metchnikovi, but that of guinea-pigs rendered by vaccination insusceptible to this organism has acquired the power of killing in a short time large numbers of the vibrio. A similar difference has been demonstrated, although less constantly, between the blood of normal guinea-pigs and that of guinea-pigs vaccinated against the cholera vibrio, which is very similar in morphology and biological properties to the vibrio of Metchnikoff. Metchnikoff attempts to break the force of these observations by the contention that the bacteria may indeed be killed by the blood-serum outside of the body, but that they are not killed by the blood and fluids in the living body. The conditions are of course different in the living body from those in the test-tube, but there is evidence that the bactericidal power of the fluids may be exerted within the body, although often less completely and less rapidly than in the test-tube.

It is permissible to suppose that antibacterial properties may pertain to the extracellular fluids within the living body, when they are not always demonstrable in fluids withdrawn from the body, and also that these properties may be latent and developed in the immune individual as the result of a reaction set up by the invasion of the bacteria ; but we have little positive evidence to support these suppositions.

It has been claimed that immunity may sometimes depend upon other properties of the fluids besides the bactericidal and antitoxic, such as simple restraint of growth or attenuation of virulence or deprivation of the power of producing toxic substances.

We have considered, thus far, the protection which may be afforded by immunizing substances when introduced into the animal body before the entrance of pathogenic micro-organisms. We have seen that as regards many important micro-organisms of disease there is no especial difficulty in conferring upon animals greater or less insusceptibility to subsequent infection. When, however, the attempt is made to give protection by these substances after the pathogenic organisms have been introduced into the body, the difficulties in attaining success become greater and greater as time elapses, and after the symptoms are well marked the difficulties are vastly greater in effecting cure than they are in simple immunization before infection.

Curative substances have been sought for among the cultural products of bacteria. Koch's tuberculin is a notable example. The specific vaccinating substances have been applied to the treatment of experimental infections. There are, however, serious obstacles to attaining success in healing with these bacterial products. These vaccinating substances require, as a rule, considerable time, two or three days, and usually longer, to produce immunity. They must therefore be obtained generally in a more concentrated form if they are to be used for acute infections. In some instances such concentration has been accomplished. This class of bacterial products, unless completely deprived of toxic properties, is likely to produce a reaction which is distinctly unfavorable, and may increase susceptibility to the micro-organism forming them. In a few instances this difficulty also has been overcome. The successes, however, which have been attained in animals with concentrated immunizing and non-toxic bacterial products injected after the entrance of the micro-organisms into the body have been only modest, and consist chiefly in conferring protection when the treatment has begun before the manifestation of distinct symptoms or in infections of slow progress and only moderate intensity.

Greater success has been reached in treating experimental infections or intoxications by means of the blood-serum, milk, or other fluids from animals which have been rendered by vaccination immune from the micro-organisms causing the disease. This method of treatment is called blood-serum therapy by Behring, who has been especially active in developing its principles. The characteristic properties of this curative serum have already been described.

The principles of blood-serum therapy have been worked out most fully for tetanus and diphtheria. It has been found that the curative properties of the serum are in direct proportion to the quantity injected and to the degree of immunity possessed by the animal yielding the serum. To produce the same curative effects in large animals as in small it is necessary to inject proportionately larger amounts, and as regards tetanus these amounts stand in approximately direct ratio to the respective weights of the animals. Of decisive influence upon the result are the stage and the intensity of the disease. If the serum be injected immediately after the reception of the virus, a larger quantity of serum is required to afford protection than a few hours previously, and the amount required increases as time elapses, until as soon as the very first symptoms of the minimal fatal dose of the tetanus poison appear in the mouse, one thousand times the quantity of serum necessary for simple immunization must be injected, and with each succeeding hour the requisite amount increases at an enormous rate, so that after twenty-four hours the necessary quantity of serum possessing an immunizing value of 1 to 1,000,000 is too great to be introduced into the animal (Behring). The larger the amount of tetanus-poison which has been received, the greater must be the quantity of healing serum injected.

A desideratum, as yet only imperfectly attained, is a method of obtaining the specific curative substances in a much more concentrated and easily preserved form than in the original fluids.

The conditions have been found, as regards progressive increase of dosage of serum according to the stage of the disease, decidedly more favorable for experimental diphtheria than for tetanus.

It is evident that the difficulties to be overcome in the successful application of serum-therapy to large animals and to man are great. It is important not to generalize, but to work out for each disease separately a sound experimental basis for this mode of treatment, as has already been done with considerable success for tetanus. This statement is sufficiently evident when we consider the varying conditions which underlie immunity from different diseases, the varying degrees of immunity attainable, and our imperfect knowledge as to both of these points as regards many diseases. The consideration of the, as yet only very limited, applications of serum-therapy to human diseases belongs to other sections of this work.

DIATHETIC DISEASES.

BY HENRY M. LYMAN.

THE term *diathesis* has been employed to designate so many different bodily conditions and diseases that it has lost a great part of its original precision, and has consequently fallen into considerable disfavor. This fact is to be regretted, for a careful analysis of the nature and conditions of disease brings clearly to view the intimate relationship that exists between a large class of bodily disorders and certain peculiarities of original constitution and function. In order to secure the advantage of such observations it is necessary to rehabilitate the term by the aid of a few precise definitions.

Before proceeding to the work of definition, it is, however, desirable to review briefly the physiological processes by which a continual circulation of matter is maintained throughout the protoplasmic molecules of a living organism. Thus viewed, a continual procession of non-living atoms is perceived to penetrate the living molecules, where they are transformed into living matter, and are subjected to various chemical reactions with oxygen by which the complex molecule is reduced once more to the condition of non-living matter that is finally expelled from the organism to give place for another group of nutrient particles. Within each protoplasmic molecule successive atoms are continually born into the world of life, where they live and act and die, and disappear to rejoin their dead companions in the universe of matter.

The continuous exchange that is thus effected within the protoplasmic molecules of an organized body constitutes the process of nutrition: it consists of assimilation, reaction, and disassimilation. Under the influence of these processes the latent energies that have been stored up in the nutrient particles that enter the organism are liberated within the molecules of living matter, and thus furnish motive-power, heat, electricity, and chemical forces which are transmitted through the intervention of the nervous system and the circulatory fluids to all parts of the body.

The controlling influence of the nervous system over the processes of nutrition is a matter of daily observation when it is made conspicuous by the effects of nervous diseases and modification of structure in the brain or the great ganglia at its base. The destruction of gray matter in the ventral horns of the spinal cord produces far-reaching and most conspicuous changes in the growth and function of distant muscles, bones, and skin. The function of nutrition may, however, be otherwise modified by causes that are connected not with any particular apparatus of the body, but with individual peculiarities of structure that characterize different ages and different sexes. Climate, food, geographical or geological location, occupation, manners and customs, hygienic surroundings, and all variations of light, heat, moisture, and atmosphere, are capable of modifying the various processes of nutrition.

Under ordinary circumstances, when the conditions of existence bear equally upon all individuals, the process of nutrition is conducted within certain normal limits which mark the normal standard of nutrition. This standard is transmitted from parent to offspring.

When, however, the individuals of a group of organized beings are numerous, and are consequently subjected to considerable variations of environment, including inequality and uncertainty in the supply of nutritive matter, considerable departure from the normal standard of nutrition soon becomes apparent. Presently the progeny of such individuals exhibit a notable deviation from the normal standard, and when such departures are sufficient to interfere prejudicially with function a pathological condition is established which either constitutes a disease or a predisposition to disease. These departures and tendencies or predispositions are subject to great variation, rendering it often difficult to draw a distinct line between states of health and states of disease.

In this connection it will be found useful to define clearly the terms "constitution," "temperament," and "diathesis," since various definitions or shades of meaning have been assigned to them all. The simplest and clearest exposition appears to be that of Bouchard. According to this eminent Parisian teacher, the term *constitution* includes all that is concerned with the structure and framework of the body, while the word *temperament* includes everything that pertains to individual characteristics of nutritive and functional activity. Since the pathological functions of the body may be performed with varying degrees of activity and intensity according to the vigor with which the processes of assimilation and disassimilation are conducted, individual temperament must necessarily vary accordingly. A strong constitution, evidenced by a firm and well-built body, is the usual foundation and substratum of an active, vivacious temperament. But a strongly constituted body may by disease or misfortune be rendered incapable of active function, so that a naturally lively temperament may be transformed into one that is dull and heavy.

Every one is endowed with a constitution and a temperament which date from the time of conception and gestation. They constitute an hereditary endowment, and under normal conditions they determine the course of nutrition during life in accordance with the normal standard of existence; but if, as a consequence of hereditary causes or of unfavorable environment, the normal processes of nutrition fail to be accomplished or are performed in a pathological manner, leading to the development of certain special diseases that are liable to appear with regularity at certain epochs or during certain periods of life, giving a peculiar character to the development and physiognomy of accidental disorders that are only indirectly connected with the function of nutrition, it is convenient to designate such a predisposition by the word *diathesis*.

This term has been frequently employed to indicate an actual condition of disease, but at present its use is limited to the indication of an existent predisposition. Obviously, a predisposition to morbid function implies a peculiar constitution and an unstable temperament; hence the definition adopted by Bouchard and his pupils, who consider a diathesis as a morbid temperament. The same school admits the existence of only two diatheses: 1, scrofula, or a predisposition to tuberculosis; 2, arthritism, or a predisposition to certain diseases characterized by retardation of the processes of nutrition.

Leaving out of view the scrofulous diathesis and restricting investigation to the arthritic diseases which possess the common characteristic of retardation in the processes of nutrition, it becomes necessary to enumerate the symptoms by which such retardation may be recognized. According to Bouchard, there are nine characteristic modes of deviation from the normal process: 1. When, after the introduction of a definite quantity of food, the return of the body to its normal weight is delayed beyond the ordinary length of time. 2. Maintenance of the standard weight of the body by less than the normal quantity of food. 3. Increase of bodily weight when only the ordinary quantity of food is taken (this does not apply to the period of growth). 4. Reduction in the amount of excreta, though the normal quantity of food be continually taken. 5. An abnormally small diminution in the weight of the body during a period of fasting. 6. Reduction below the normal quantity of excreta during a period of fasting. 7. Appearance in the excreta of incompletely oxidized substances like uric acid, oxalic acid, or other organic acids, and the volatile fatty acids. 8. Excessive accumulation in the body of various proximate principles when the diet is of a normal character. 9. Abnormal reduction of the temperature of the body during periods of repose, abstinence, and sleep.

These departures from the normal standard of nutrition can be rarely encountered at the same time in the same individual; but in many instances the existence of one or more clearly defined deviations from the physiological standard may be discovered, furnishing invaluable indications regarding the pathological condition of the patient. The occurrence of such morbid variations is usually observed in certain families and in certain individuals, who from infancy to advanced age exhibit now one now another mode of perversion, constituting a group of successive pathological experiences that are connected together by the common fact of retarded nutrition. This fact constitutes a definite predisposition or diathesis. The arthritic diathesis, therefore, consists in a predisposition to arthritic diseases, a group of disorders which take their generic name from the fact that they usually occur among individuals who are subject to gout or rheumatism—articular diseases which were anciently confounded together under the common term arthritis.

The principal arthritic diseases are biliary lithiasis, gravel, obesity, diabetes, gout, acute articular rheumatism, the different varieties of chronic rheumatism, and abarticular rheumatism—a comprehensive term that includes muscular rheumatism, asthma, recurrent coryza, sibilant bronchitis, acute emphysema, acid dyspepsia, eczema, urticaria, hemicrania, and those forms of neuralgia that exhibit a rheumatic type. A condition that is exceedingly common either as a preliminary or as a concomitant of these disorders is designated as the acid dyscrasia. It is one of the prominent features of the arthritic diathesis.

THE ACID DYSCRASIA.

BY HENRY M. LYMAN.

DURING the processes of oxidation in the tissues a considerable number of organic acids are produced. The direct decomposition of nitrogenous protoplasm yields certain nitrogenous acids—*e. g.* uric, hippuric, and oxaluric acids. Of these, the first and the last may be still further transformed into oxalic acid. Certain saccharine derivatives are also formed which yield lactic acid or its derivatives; substances of the nature of cholesterin and the volatile fatty acids make their appearance—*e. g.* caprylic, caproic, valerianic, butyric, propionic, acetic, and oxalic acids.

The oxidation of fats results in the formation of stearic, oleic, and palmitic acids, and it may also yield the volatile fatty acids.

From starchy substances are derived lactic, butyric, acetic, and oxalic acids.

In the stomach the transformation of saccharine substances may yield lactic, butyric, propionic, acetic, and formic acids. In the intestines caproic acid may be produced by the action of the pancreatic juice upon peptones, acetic acid may be derived from glycocoll, and valerianic acid can be in like manner obtained from the pancreatic juice. From these different sources it is evident that an excessive quantity of acids may exist in the alimentary canal, and may be transferred by absorption to the circulating liquids of the body and to the tissues, where they undergo more or less complete transformation and elimination through the different excretory organs. Of these organic acids, carbonic acid and uric acid do not by their presence increase the acidity of the tissues, since carbonic acid never exists in a state of freedom, but is always combined with bases for which it manifests an affinity; and uric acid, if uncombined with bases, is rapidly eliminated through the kidneys and does not manifest acid qualities.

Of those acids which are not decomposed by oxidation during their passage through the tissues, formic acid, acetic acid, butyric acid, and several other volatile fatty acids are eliminated by the skin. Butyric acid and cholalic acid are eliminated with the fæces, while the urine serves as a vehicle for the discharge of uric, hippuric, oxaluric, carbonic, taurylic, damaluric, damalic, succinic, and oxalic acids.

While it is true that the gastric juice may under certain conditions contain an excess of hydrochloric acid, yet in the majority of cases that are characterized by excessive acidity of the stomach the condition is due to the formation of lactic, acetic, and butyric acids as a consequence of fermentation induced by the presence of microphytic ferments in the gastric contents. Such fermentation leads to the passage of acid liquids into the intestinal canal, where there is no adequate provision for the proper neutralization of these organic acids. The intestinal mucous membrane therefore becomes irritated and inflamed to a degree that seriously interferes with the process of intestinal digestion. It is in this way that the acid diarrhœa of young children is excited.

The lactic acid that is absorbed from the alimentary canal produces a deleterious influence upon the tissues of the body. It renders soluble the calcareous salts that should give stability to the bones, and leads to the development of diseases like osteomalacia and rickets. It is not improbable that renal and cutaneous irritation may be in like manner excited during the elimination of lactic acid through the glandular structures of the kidneys and of the skin. Certain cutaneous diseases may be thus excited or aggravated, and the urine may become clouded by an excessive discharge of urates and oxalates, laying the foundation for renal and vesical calculi.

The acid state of the perspiration in certain diseases that are accompanied by copious sweating is undoubtedly due to the excessive presence of various organic acids in the liquids and solids of the body. The peculiar odor that is exhaled from the surface of the body in certain disordered conditions of health is due to the elimination of volatile acids through the skin. The dependence of such conditions upon imperfect oxidation is rendered probable by the fact that active exercise, a febrile condition, and the administration of remedies that accelerate oxidation are followed by the reduction or total disappearance of the offensive exhalations.

A conspicuous example of the consequences of excessive production or accumulation of an organic acid in the body is furnished by the complex of symptoms that is developed when oxalic acid exceeds the normal quantity in the blood and in the tissues. A litre of healthy urine should not contain more than 0.02 gramme of oxalic acid, but in certain conditions of ill-health this quantity is greatly exceeded. Under normal conditions oxalic acid combines with calcic oxide, and, held in solution by phosphate of sodium, is eliminated harmlessly with the urine. Certain vegetables and fruits, especially the pie-plant, contain the substance, and their use as food causes a notable augmentation of the oxalate of calcium in the urine. It exists in the blood of the victims of gout, and among patients who are scrofulous, tuberculous, hypochondriacal, obese, and neurasthenic. Such patients are always tired. They perspire upon the slightest provocation; their muscles are enfeebled; their nervous system is excessively irritable; they are unrefreshed by sleep at night; and they experience an uncontrollable somnolence during the middle of the day. In many cases the breath is offensive and the fæces are acid. Sometimes tremor is witnessed in the limbs; emaciation is often rapid by reason of the solution and removal of calcium and phosphorus from the framework of the cells of the tissues. The tricalcic phosphate of the tissues surrenders two equivalents of calcium, which enter into combination with oxalic acid, while the remaining acid phosphate of calcium passes in a state of solution out of the tissues and is eliminated with the urine.

The palliative treatment of oxaluria consists in the administration of alkaline salts, especially the bicarbonate of sodium or of potassium. Other organic salts of these bases—*e. g.* the citrate, tartrate, and benzoates—may also be prescribed. Calcic salts should not be administered, since their combinations are insoluble and tend to the formation of urinary calculi. The sodic and potassic salts should be given in moderate doses, not exceeding 40 to 80 grains a day, for a period of ten days during each month. Bouchard has observed that after a long-continued administration of small doses of soda the earthy phosphates make their appearance in neutral or alkaline urine.

In order to repair the loss of phosphates, eggs, fish, and cereal foods which are rich in phosphorus should be furnished in abundance. The alkaline phosphates of sodium and of potassium are also useful, but the phosphate of calcium should be avoided.

Excessive acidity of the stomach may be temporarily relieved by the administration of various alkaline solutions. Dyspeptic conditions should be combated with dilute hydrochloric acid or other mineral acids.

But the most efficient remedies are those which increase the processes of oxidation in the tissues. For this purpose the patient should be made to take exercise in the open air, with special reference to the promotion of muscular movement in the upper extremities and thoracic walls, so as to favor respiration and the introduction of oxygen into the blood. Rowing and gymnastic exercises are admirably adapted to this end. For those who cannot go abroad massage should be recommended, and the function of the skin should be stimulated by friction, cold sponging, and the use of salted towels, brine baths, hydropathic methods, etc. Mountaineering, voyaging, hunting, fishing, and an active life in the open air may be relied upon to complete the cure.

RICKETS.

By HENRY M. LYMAN

Definition.—Rickets is a disease of infancy and early childhood, characterized by excessive proliferation of the osseous tissues, with an insufficient calcification of the growing bones. As a consequence of this morbid process the bones of the skeleton are softened, and, yielding to external pressure, become deformed. It is supposed that the name "rickets" is an English modification of the old Norman word *riquets,* which signifies a humpback.

SYNONYMS.—Rachitis; Rachitismus; Morbus anglicus; Zweiwuchs; Doppelte Glieder; Rachitisme.

Etiology.—The actual cause of rickets is yet unknown, and the knowledge of its etiology is at present limited to the conditions that appear to favor its development.

Rickets is a disease of the period of first dentition, apparently by reason of the fact that the effects of improper feeding during the period of early infancy are then most conspicuous.

The majority of observers are of the opinion that the children of rachitic parents are, by that fact, more than others predisposed to the disease. There is considerable difference of opinion among pathologists regarding the relationship of rickets with scrofula and tuberculosis. Eminent authorities have expressed the belief that there is an actual antagonism between these diseases, while others are of the opinion that the signs of rickets may be always detected among children who have died from other chronic diseases. A similar connection between syphilis and rickets has also been affirmed and as strenuously denied.

The geographical distribution of rickets is a matter of interest. The disease exists chiefly in the lower quarters of large cities in the temperate zone. It is rare in the tropics, probably on account of the free exposure of children in the open air and sunlight of warm climates. It is seldom observed in elevated mountainous regions, probably because large cities with their squalid poverty do not exist in such regions. It is in the miserable homes of the poor, in cold, damp, and dark tenement-houses, that the disease is most frequently encountered, though its minor forms are not uncommon among the children of the better classes who are brought up by hand through failure of the mammary secretion on the part of debilitated mothers. Among such children the effects of premature weaning and the replacement of mother's milk by a mixture of farinaceous substances and warm water result in the production of chronic indigestion that is characterized by excessive acidity throughout the alimentary canal; the blood becomes overcharged with the acids that are absorbed from the intestine, and the ossification of the bones is consequently disturbed. On the other hand, when children are retained at the mother's breast beyond the expiration of the first year of life the milk affords insufficient nutriment for the growing tissues, and disorder may be thus excited.

It has been shown that an excess of lactic acid in the stomach and an insufficiency of alkali in the intestines, either as a consequence of fermentation or of deficient biliary secretion, will hinder the assimilation of the phosphates of calcium that are needed for the healthy nutrition of the bones. Now, it has been observed that in the majority of rickety children the perspiration, urine, and fæces are characterized by excessive acidity. It is this acid dyscrasia that most seriously interferes with the normal nutrition and healthy ossification of the growing skeleton. It is not improbable that the luxuriant growth of morbid tissues in the diseased bones is, in part at least, due to the fact that the process of cell-proliferation is not restrained by the normal deposition of calcareous salts. Many things, however, in this connection, remain yet unexplained.

Pathological Anatomy.—The normal growth of healthy bones proceeds simultaneously in the cartilaginous epiphysis, the periosteum, and the medullary portion of the shaft of the bone. In the normal condition the epiphyseal cartilage is separated from the bony shaft by a transverse layer of chondroid substance. It is in this layer that the process of growth is chiefly effected by the multiplication of cartilage-cells and by the formation of osteoblastic cells, which are finally transformed into osteoplastic cells, in which the solidifying calcareous salts are deposited.

In a bone that is undergoing rachitic disease the epiphyseal cartilage is separated from the medullary cavity by a band of chondroid substance an inch or more in length. This is gray and soft, and bounded at either extremity by an irregular and jagged margin, especially upon the side toward the shaft of the bone. Next to this layer is a band of red spongy substance that resembles a bone that has been partially softened by the action of an acid. Between this and the medullary canal of the bone intervenes the originally ossified substance, now reduced to a condition of disease by a process of decalcification.

Histological examination shows that in the chondroid layer the proliferation of cartilage-cells is greatly exaggerated, and calcification proceeds in an irregular manner. The spongy layer resembles a fine sponge or a fragment of bone from which the calcareous salts have been partly removed by hydrochloric acid. The spaces in the spongy framework are filled with a soft and reddened jelly that consists of round and angular cells and numerous blood-corpuscles, constituting what is by some authors considered as modified cartilage partially infiltrated with mineral matter, while by others it is thought to be an imperfectly developed osseous tissue.

The process of ossification beneath the periosteum that covers the shaft of the bone exhibits morbid changes similar to those which invade its extremity. The morbid process results in subperiosteal thickening, which is most conspicuous about the middle portion of the shaft, giving a spindle shape to the long bones of the body which have been thus diseased. By the encroachment of the thickened mass the central canal of the bone is frequently constricted. The increased vascularity of the diseased tissue gives it an appearance somewhat resembling the pulp of the spleen, which accounts for the belief formerly entertained that it was of the nature of a hæmorrhagic exudation. In the later stages of the disease the morbid growth becomes condensed and closely connected with the periosteum, forming a thickened and ossified structure that is much more voluminous than the normal bony shaft. Similar changes are manifested within the medullary canal.

In an advanced stage of rickets islets of the original bone may be discovered in the midst of the rachitic tissues. These remnants of the original process of ossification may be recognized by the direction of their lamellæ, parallel with the axis of the bone instead of being perpendicular or obliquely placed as in the rachitic substance. The vascularity of the diseased portions of the bone is much greater than normal, so that the mineral matters originally deposited are rapidly dissolved and removed, leaving a yielding substance that is readily bent out of shape by the pressure to which it is exposed in bearing the weight of the body. Similar changes take place in the flat bones of the skeleton. Chemical analysis reveals the fact that not less than two-thirds of the mineral constituents of a rickety bone are removed during the period of disease. But with its termination and cure the spongy tissues are sometimes infiltrated with mineral matter without normal ossification, so that the diseased epiphysis may become eburnated, exhibiting a hardness like that of ivory, and effectually arresting the longitudinal growth of the shaft of the bone. In the majority of cases, however, the process of recovery consists in a gradual absorption of the spongy substance and a more or less complete return to the normal type of ossification.

When the rachitic bone is fractured the solution of continuity takes place within the periosteum, constituting a bending rather than a breaking of the softened tissues, like what may be observed when a green stick is bent at an angle within its sheath of bark.

Symptoms.—Before the appearance of osseous change and deformity there is a period of invasion that is characterized by gastro-intestinal disorders, diarrhœa, night-sweats, debility, and a moderate degree of fever. The digestive disorders are characterized by frequent vomiting, diarrhœa alternating with constipation, the stools being excessively acid and mingled with undigested food. A similar excess of acid is present in the perspiration, which flows copiously from the abdominal surface and from the scalp, so that during sleep the pillow upon which the child's head is supported becomes saturated with moisture.

The first evidences of deformity are usually visible about the wrists and ankles, and then in the condyles of the femurs. The anterior extremities of the ribs also manifest enlargement. Sometimes the disorder is restricted to these epiphyseal enlargements, but in other cases the shafts of the bones are bent and incurved by the traction of the muscles or by the weight of the body or by atmospheric pressure. In the later stages of the disease deformity is sometimes caused through arrest of the longitudinal growth of the bones by reason of eburnation of the epiphyses. When the spinal column and the thoracic skeleton are involved, great deformity of the trunk results from antero-posterior and lateral curvatures of the spine, accompanied by flattening of the thorax and lateral deviation of the ribs; the pelvis also becomes deformed, and the cranium exhibits flattening of the occipital portion, together with prominence of the frontal centres of ossification, and an increase of the biparietal diameter, giving to the whole head a peculiar, square, and box-like appearance. The angles of the jaw sometimes exhibit excessive squareness and prominence, and there may be great vaulting of the roof of the mouth, with protrusion of the narrowed maxillæ, consequent upon yielding of the softened bones during the act of suction on the part of the nursing infant. In a large proportion of cases of rickets the disease is most conspicuous in the lower extremities. The process of deformity seems to injure the ankles and legs more than the thighs, and the lower portion of the body more than its upper portion, while the distal portions of the upper extremities suffer in preference to the upper portion of the skeleton and the skull. To this rule there are, however, many exceptions. Curvature of the spine and deformity of the thorax, without any corresponding changes in the pelvis and lower extremities, are not infrequent.

Deformity of the cranium is the result of belated closure of the fontanelles, which, instead of disappearing at the end of the second year of life, sometimes remain open until the third or fourth year. As a consequence the brain, continuing to grow, dilates the cranium until its dimensions are exaggerated out of all proportion to the bones of the face. There is a certain resemblance between the form of the rachitic skull and that of the hydrocephalic cranium, but the intellectual condition in the two diseases is entirely different. The hydrocephalic brain is imperfectly developed, and is overwhelmed by a dropsical transudation that interferes with intellectual activity, but the rachitic brain, not retarded by compression, is free to grow, and its functions are unimpeded and often somewhat extraordinary. The intellectual vivacity and incisive wit of rachitic humpbacks have always been remarked, and it was from this class that the court fools and privileged jesters of the mediæval princes and monarchs were recruited.

As a consequence of the deformity of the jaws above mentioned the process of dentition may be seriously disturbed. Great delay in the eruption of the first teeth is an early sign of infantile rickets. By reason of the narrowing of the jaws the teeth are often crowded out of place, and present great irregularity in their position and order of appearance. The form of the teeth is also modified, so that they appear too large and rudely fashioned. The enamel, instead of gradually thinning out upon the neck of the tooth, sometimes terminates abruptly in a ridge between the neck and the crown, and upon the incisor teeth the enamelled surface often appears roughened, as if eroded by an acid.

Deformity of the thorax is the most frequent and conspicuous of the consequences of rickets. The normal antero-posterior and lateral curves of the spinal column are sometimes greatly exaggerated, and the dorsal lateral curvature has its convexity always directed toward the right side of the trunk. A compensatory curve in the opposite direction is developed in the lumbar region. The spine is also twisted upon itself, so that the ribs project strongly upon the side of the lateral convexity of the spine, while there is a corresponding depression of the ribs upon the opposite side of the trunk. The posterior line of incurvation of the projecting ribs forms the prominent ridge of the humpback, and a corresponding projection of the costal cartilages and angularly curved ribs exists in front upon the opposite flattened side of the thorax. At the point of junction between the costal cartilages and the anterior extremities of the ribs an ossified protuberance can be felt beneath the skin upon each rib, distinctly marking the line of morbid proliferation and ossification in the sterno-costal epiphyses. The sternum itself is often pushed forward and rendered prominent in a way that gives to the laterally-compressed thorax a remote resemblance to the thoracic structures of a bird; hence the term *pigeon-breasted* that is used to designate this deformity. The lower portion of the thoracic walls is often considerably everted in consequence of the compression of the lateral walls of the chest by the weight of the atmosphere, while the abdominal viscera, pressing upward and outward, expand the lower ribs, which rest upon the diaphragm. Excessive enlargement of the abdomen, consequent upon gaseous distension of the stomach and intestines, together with the downward displacement of the liver that is dependent upon thoracic deformity, constitutes a not uncommon symptom of rickets.

The pelvis sometimes exhibits deformities that are due to pressure upon the bones. Its lateral walls are sometimes pressed inward, causing anterior projection of the symphysis pubis; but, in those cases in which there are lateral curvature of the spinal column and unequal distribution of the weight of the body upon the sides of the pelvic skeleton, its wall is flattened upon the same side with the deviation of the lumbar spine, while a corresponding projection of the bony structures at the inferior strait exists upon the opposite side.

As a consequence of thoracic deformity the lungs are of unequal size and do not reach their normal growth. The heart is often displaced upward or to the right, and, owing to the fact that it is forcibly crowded against the anterior wall of the thorax, its pulsations are unusually conspicuous: sometimes the difficulty that attends its movements leads to actual hypertrophy of its muscular structure. Emphysema is not uncommon among rachitic subjects, and diseases of the respiratory organs are especially dangerous by reason of the difficulties that attend the movement of the lungs and the pulmonary circulation of the blood. Special tendency to spasmodic contraction of the laryngeal muscles is not uncommon among rickety children, who consequently often manifest the symptoms of spasmodic croup, laryngismus stridulus, spasm of the glottis, and exaggerated forms of whooping cough.

Pelvic deformity is greatly to be feared in female children, since it will render parturition difficult if not altogether impossible.

Locomotion is rendered laborious and ungraceful when the extremities are deformed by rachitic curvature of the long bones. Knock-knees, bow-legs, and club-feet in every degree of variation result from deformities that are thus originated.

In certain cases of rickets the bones present such fragility that they are very easily broken, and thus add to the deformity of the skeleton by imperfect union of the fragments or by the establishment of false joints when ossification of the callus does not take place.

In certain exceptional cases the unborn fœtus is affected with deformity of the long bones of the extremities, or with multiple fractures, enlargement of the fontanelles, and other modifications of the skeleton that indicate the existence of intra-uterine rickets. It is also probable that in many cases the children of ill-nourished and exhausted mothers manifest at birth the symptoms of congenital rickets. This fact should be remembered when caring for the health of pregnant women.

The commencement of rickets is occasionally delayed until the later years of childhood. The disease then appears as a sequel of an acute infective malady like measles, and it is usually accompanied by the symptoms of anæmia and cachectic albuminuria. It is probable that the lateral curvature of the spine that is sometimes observed at the age of puberty is a belated symptom of rickets.

Opinions are divided regarding the existence of rickets in an acute form. A number of observers have noted cases in which acute swelling of the limbs, accompanied by fever and the general symptoms of inflammation, were followed by the development of a characteristic enlargement and deformity of the epiphyseal extremities of the bones. Other excellent observers, however, believe that these cases should be referred to osteomyelitis or periostitis, or to purpura with subperiosteal hæmorrhage, and especially to scurvy.

Diagnosis.—In fully-developed cases of rickets the diagnosis is not difficult. Latent forms of the disease may be suspected when the teeth appear irregularly and when their eruption is unusually delayed. Excessive perspiration upon the scalp and obstinate diarrhœa afford valuable indications of the disease. It cannot be confounded with osteomalacia, since that occurs only among adults, and is not accompanied by those pseudo-inflammatory conditions of the bones which are encountered during the active stage of rickets. Congenital syphilis sometimes exhibits a diseased condition and separation of the epiphyses, but this incident occurs during the earliest period of life and is accompanied by other symptoms of hereditary syphilis. From chronic hydrocephalus rickets may be distinguished by the fact that convulsions frequently occur, and intellectual hebetude is always present as a consequence of the dropsical effusion.

Prognosis.—Uncomplicated rickets usually admits of a favorable prognosis, especially when favorable hygienic conditions can be secured. It is true that various deformities will probably persist as evidence of the early experience of disease, though a certain amount of incurvation of the long bones may spontaneously disappear with the return of health. The occurrence of considerably deformity in the thoracic portion of the skeleton predisposes the individual to pulmonary and cardiac disorders. Pelvic deformity will render the act of parturition dangerous for the adult female.

Treatment.—The treatment of rickets demands the most careful regulation of the mode of life, diet, and medication of the patient. If possible the child should be removed from a damp and ill-ventilated home to dry and airy apartments. If this cannot be accomplished, the parents should be instructed how to secure the best ventilation and the greatest amount of pure air and sunshine in their home. Rickety children should be made to spend as much time as possible in the open air, even during cold weather. The bed upon which the rickety infant lies should not be made of feathers, but must consist of some firmer material—preferably a hair mattress and pillow. Frequent baths are desirable, especially sponge baths containing salt. The skin should be rubbed dry and must be well protected with flannel. If unable to sit up, the position of the body should be frequently changed, in order to prevent the effects of pressure upon yielding bones.

As far as possible the food of the child during the first year of his life should consist of mother's milk, but if that cannot be procured the child should be fed with cow's milk. Unless such milk can be obtained from a cow that is known to be healthy, it is better in large cities to make use of the milk that is furnished wholesale from country dairies, since the mixture of the milk of a tubercular cow with the milk of many other healthy animals neutralizes the danger of tubercular infection from that source. Fresh milk from a healthy animal is, however, the best substitute for mother's milk that can be obtained. It should be sterilized as soon as drawn, and may be safely preserved for a considerable time in the sterilizing apparatus, which may be obtained from any considerable dealer in medical and surgical appliances. If the financial condition of the family does not permit such an expenditure, it will be necessary to instruct the mother to prepare the milk that is furnished for her babe by heating it to near the boiling-point as soon as procured, and by adding a little bicarbonate of sodium to the liquid, which must then be kept in a bottle that is closed with a cotton plug. Since the quantity of casein in cow's milk is considerably larger than in human milk, it is necessary to dilute it by the addition of water: in order to prevent the formation of solid curds in the stomach, it is advisable to make use of barley-water as the diluent or of oatmeal gruel if the patient suffers from constipation. Barley-water and oatmeal gruel may be prepared by boiling two or three teaspoonfuls of barley or of oatmeal in a pint of water until thoroughly softened. A pinch of salt should then be added to the decoction, which must be carefully strained to remove the grains and grits. Enough milk-sugar, or loaf-sugar if the first cannot be obtained, to make the mixture palatable should be added to the liquid, which may be then used to dilute the milk in the ratio of four parts to one, during the first weeks of life. As the child grows older the quantity of milk should be increased, until at six months it equals the quantity of the diluent. With advancing age the quantity of milk should be progressively increased, until at the end of the year fresh milk can be given without dilution. The importance of weaning the child at the proper time has been already emphasized on a previous page. Cow's milk and cream should still constitute the principal articles of diet. Stale bread, especially that which is made from whole-meal flour, may be added in the form of bread and milk. But the use of farinaceous food should be gradually introduced. Animal food must still form the greater part of the dietary. Beef tea, mutton broth, Valentine's extract of beef, meat pulp obtained by scraping raw beef, should all be allowed with considerable freedom. If the child object to the taste of raw-meat pulp, the substance may be advantageously mixed with currant-jelly. A raw egg beaten up with milk and rendered palatable with a little sugar is a valuable article of food for somewhat older children, and in cases complicated with diarrhœa koumyss or matzoon will be found invaluable. Maltine and other preparations of malt, when they do not add to the acidity of the alimentary canal by fermentation, may be allowed. Cod-liver oil of the purest and most tasteless quality should be given three times a day in doses of a quarter of a teaspoonful and upward, according to the age of the patient.

The pharmaceutical treatment of rickets consists largely in the administration of phosphorus. This should be given dissolved in cod-liver oil, so that the daily ration shall contain from $\frac{1}{150}$ to $\frac{1}{100}$ grain, according to the age of the child. This amount may be given in divided doses each day.

The tendency to excessive acidity in the contents of the alimentary canal should be corrected by the administration of the bicarbonates of sodium or potassium. An excellent preparation for this purpose, and for the regulation of the bowels when a tendency to constipation or diarrhœa exists, is furnished in the form of the "neutralizing cordial," the compound syrup of rhubarb and potassium. This compound serves to neutralize any excess of acid, and it also acts as a tonic to the gastro-intestinal mucous membrane and as a stimulant to healthy secretion on the part of their glandular structures. It should be given in doses varying according to the age of the child, from fifteen drops to a teaspoonful, two or three hours after each meal. Besides its therapeutic qualities, it possesses the virtue of being quite palatable, so that by the majority of children it is welcomed as an agreeable medicine.

For the relief of anæmia iron, in the form of the syrup of the iodide of iron, should be given, or the saccharated carbonate of iron may be given in appropriate doses. When the bowels are constipated minute doses of nux vomica and podophyllin may be given. Gentian is also recommended, but the difficulties that attend the administration of such bitter tonics will often tax the ingenuity of the physician. The parvules and granules and tablets that are now furnished by the manufacturing chemists will often afford a convenient solution of the difficulty, but among the children of the poor summary measures will generally be necessary in order to secure efficient medication. Many physicians recommend the use of the lacto-phosphate of calcium and other calcic salts. But since the ingredients for the formation of healthy bone-tissue are present in milk and in the other articles of food that have been already recommended, it is better to secure their digestion and assimilation rather than to dose the patient with what may, after all, result in a deleterious excess of lime in the food.

The occurrence of diarrhœa in connection with rickets requires the ordinary pharmaceutical treatment with considerable doses of the subnitrate of bismuth. Bronchial and pulmonary complications must be treated in accordance with the general rules that govern the management of such disorders. Spasm of the glottis and other convulsive phenomena require the administration of the bromide of potassium or sodium. Inhalation of chloroform may be occasionally necessary in sudden paroxysms of the disease, but ordinarily a few grains of chloral administered at proper intervals will be sufficient, in connection with the use of bromide of sodium, to ward off a severe attack. In all cases that are characterized by spasmodic tendencies the causes of peripheral irritation should be sought and removed if possible. The eruption of each tooth is often preceded in such cases by convulsions or by respiratory spasms. Lancing of the gums frequently affords considerable relief.

The deformities that result from rickets may sometimes be relieved by proper gymnastic exercises, but when they are extensive and severe orthopædic apparatus and surgical treatment are often needful. Great discretion is necessary in the application of apparatus to the lower limbs, since everything of a weighty and clumsy character will be found to interfere with the active movements of the growing child, and may thus do more harm than good.

OSTEOMALACIA.

BY HENRY M. LYMAN.

Definition.—Osteomalacia is characterized by decalcification and softening of the bones. It is a disease that occurs during adult life and old age, differing in this respect from rickets, which is manifested only during early life as an aberration of the formative processes by which the skeleton is developed. Osteomalacia, on the contrary, destroys the solidity of bones that have been already matured and are far advanced in age.

SYNONYMS.—Mollities ossium ; Malacosteon.

Etiology.—In its most conspicuous forms osteomalacia is a rare disease; its minor manifestations are not uncommon. It occurs among women more frequently than among men, in the ratio of 8 to 1. In fact, the principal causes of osteomalacia are connected with the functions of pregnancy and lactation in the majority of cases. In one-half of the patients the commencement of the disease was noticed during pregnancy, and of the remaining female patients one-half became affected as a consequence of long-continued or frequent lactation.

Of the cases that cannot be referred to the generative functions the majority are developed as a consequence of senility. While the disease as it occurs during pregnancy and lactation appears to be the result of abstraction of the calcareous salts from the bones for the benefit of the growing fœtus or infant, a similar removal of the calcareous elements of the skeleton during old age, when the functions of nutrition are enfeebled, may be ascribed to unfavorable hygienic surroundings, insufficient diet, and the use of drinking-water that contains an insufficient amount of calcareous salts.

Symptoms.—Osteomalacia is usually developed in an insidious manner, and is characterized by the occurrence of ill-defined pain in the vertebral column and pelvis, where the disease is usually located. The pain is continuous and deep-seated, without implication of the muscles, nor is it increased by muscular movement like the pains of rheumatism. It is aggravated by the warmth of the bed and by violent manipulation or percussion over the affected region. Sometimes it assumes a paroxysmal character, but usually it is constant, though aggravated by long-continued confinement in one position.

When the disease is connected with pregnancy, pain is most severe in the pelvic portion of the body, especially in the neighborhood of the ischia ; but in elderly patients the seat of pain is ordinarily located in the spinal column and in the ribs. It is aggravated by the erect position, by walking, and by stooping toward the ground.

After an introductory period of invasion that is more or less painful, the affected bones begin to exhibit signs of softening and deformity. Among puerperal women the pelvis is chiefly affected. The superior and inferior straits become narrowed through inward pressure of the lateral portions of the bony wall of the pelvic cavity, together with forward protrusion of the promontory of the sacrum and of the symphysis pubis. Parturition is greatly impeded, if not rendered impossible, by this deformity, and sometimes the functions of the bladder and rectum are also hindered.

Among old people osteomalacia causes a progressive reduction in the height of the patient by reason of yielding and shortening of the vertebræ. The spinal column is bent antero-posteriorly or laterally, causing various deformities of the thoracic skeleton, in this respect producing modifications of the form like those which result from rickets.

The bones of the head are rarely affected, but those of the limbs may exhibit softening and deformity. The distal phalanges of the toes and of the fingers sometimes exhibit morbid enlargement.

A very characteristic feature in connection with osteomalacia is the occurrence of great fragility in the bones of the skeleton, so that fractures frequently occur as a consequence of insignificant causes that are quite out of proportion with the magnitude of their effects. In this way fractures of the ribs have been produced by the muscular effort of coughing, and other bones have been known to give way during the act of turning over in bed.

The movements of respiration and of the heart are sometimes considerably hindered by the resultant deformity of the thoracic wall. The functions of digestion are always disturbed, but this condition must be regarded rather as a cause than as an effect of the disease. Chronic catarrhal inflammation of the gastro-intestinal and bronchial mucous membranes are usual occurrences. Albuminuria is also sometimes observed.

Pathological Anatomy.—The changes that are determined in the skeleton by the development of osteomalacia consist in the simple abstraction of calcareous salts from the osseous tissue. The marrow of the bones becomes at first intensely vascular, and its cellular elements are considerably multiplied: during the later stages of the disease they undergo fatty degeneration. The fundamental substance of the bones, after it has given up its calcareous salts, exhibits a fibrillary structure and becomes more or less translucent: it will no longer yield gelatin when boiled.

An excess of lactic acid is frequently present in the diseased bones, and the alkalinity of the blood is considerably diminished. The urine also contains lactic acid, but the calcareous salts are not increased in that excretion. Sometimes calculous masses of carbonate and phosphate of calcium and magnesium have been found in the urinary organs, but the greater portion of the salts that are removed from the bones are eliminated through other channels. During pregnancy and lactation they are deposited in the tissues of the fœtus or are conveyed through the milk into the body of the growing infant.

Pathology.—It is under the influence of the excessive presence of lactic acid in the bones that the calcareous salts are dissolved and removed from the tissue in which they had been normally deposited. The amount of lactic acid in a healthy condition of the body can never reach deleterious proportions, but when the process of oxidation is retarded the acids that are continually being formed are no longer oxidized into harmless substances, and they accumulate in the bones, with a result that is prejudicial to the integrity of the skeleton.

An intimate relationship between diabetes and osteomalacia has been sometimes observed. Osteomalacia occurring under unfavorable hygienic surroundings is sometimes exchanged for diabetes when the patient is placed under more favorable conditions of diet and environment. It may be that under such circumstances there is at first a transformation of sugar into lactic acid, which by its presence excites the symptoms of osteomalacia, while at a later period, when the conditions no longer promote such transformation, the symptoms of osseous disease subside and glycosuria appears. It is highly probable that the phosphaturia accompanied by osseous pain that occurs in many cases of pulmonary consumption may be due to the development of an acid dyscrasia as a consequence of digestive disorders and retarded nutrition consequent upon tubercular disease. In such cases the solution and removal of phosphatic salts seldom proceeds far enough to admit of softening and deformity of the bones. But in this and in other unwholesome conditions of existence the impairment of nutrition may be indicated by excessive fragility of the bones which have been deprived of a portion of their mineral constituents. In such pathological conditions not only the mineral salts, but also the nitrogenous and fatty constituents of the bones, are considerably diminished and replaced by water. These changes are not identical with the ordinary modifications of structure which are common characteristics of extreme age: they appear to be the specific results of imperfect nutrition, insufficient oxidation, and unfavorable hygienic surroundings, including exposure to cold and damp in the abodes of poverty.

Diagnosis.—During the early stage of osteomalacia painful symptoms have been erroneously referred to rheumatism or to neuralgia, but when deformity of the skeleton exists without any history of rickets in early life, the diagnosis is not difficult.

Prognosis.—Recovery is almost unknown. The course of the disease is slow and interrupted by occasional remissions. Sometimes death occurs in less than a year after the appearance of the first symptoms, but life may be prolonged for a number of years, though rarely for ten years. Successive pregnancies exert an unfavorable influence upon the progress of the disease. Death usually results either from exhaustion or from intercurrent pulmonary diseases, bed-sores, and other consequences of prolonged confinement in a condition of helplessness.

Treatment.—The environment of the patient usually demands improvement; digestion must be invigorated by cold baths, friction of the surface, gentle massage, and exercise in the open air. The food should be chosen with reference to an abundant supply of phosphates and other salts that are needed for the nutrition of the osseous tissues. Milk, eggs, and whole-meal flour are to be preferred, and are probably more efficacious than the preparations of phosphorus, iron, quinine, cod-liver oil, etc. that are usually prescribed.

The frequent dependence of the disease upon activity of the generative functions has suggested the removal of the ovaries with a view to the prevention of pregnancy and lactation. This operation and the operation of Porro have been in a number of instances followed by an arrest and cure of the disease.

OBESITY.

BY HENRY M. LYMAN.

Definition.—Obesity is characterized by an excessive accumulation of fat in the adipose tissue, hindering, and finally rendering impossible, the functions of the various organs of the body. The distribution of this accumulation of fat is seldom uniform, being sometimes excessive in one part of the body and sometimes in another. Particular organs may be greatly encumbered, while others experience very little impediment. But there is a close relationship between the condition of the internal cavities and that of the subcutaneous areolar tissue, so that an extensive accumulation of fat beneath the skin may be accepted as sufficient evidence of a similar internal burden.

The heart is the principal sufferer from an excessive deposit of fat within the visceral cavities. Adipose tissue is increased under the visceral pericardium between the ventricles of the organ and around the origin of the large blood-vessels. During the later stages of the disease the connective tissue of the ventricles becomes infiltrated with fat. This is especially conspicuous in the left ventricle, the muscular fibres of which are separated from each other by the redundant adipose tissue. The nutrition of the muscular fibres is thus seriously affected, and the muscular substance passes into a condition of atrophy and fatty degeneration, interfering with the movements of the heart through the destruction of its contractile substance and the additional labor that is necessary to overcome the weight of the external mass of fat.

The liver is often involved in a somewhat similar manner, but it is less exposed to pressure from external accumulations of adipose tissue. It is within the hepatic cells that the process of infiltration chiefly interferes with the functional activity of the organ.

Under normal physiological conditions of nutrition the adipose tissue of the body stores up only that amount of fat which is needed for the continuous supply of fatty matter to the tissues of the body in the absence of a sufficient quantity of such nutriment from the daily food that is supplied to the individual. Adipose tissue is present or may be formed wherever there is connective tissue, except in the lungs, spleen, sheath of the penis, and in the brain. In the normal condition it forms about one-twentieth of the total weight of the body.

The physiological store of fat is normally recruited from the fats that enter the circulation either in the form of an emulsion or in derivative combinations, forming glycero-phosphoric acid, fatty acids, and soaps. A portion of the fat is held in solution by the soaps that are formed through the action of fatty acids upon alkaline bases in the small intestines. Glycerin and the fatty acids are produced by the action of pancreatic juice and bile upon fat. The liberated glycerin enters into combination with phosphoric acid that has been displaced from the alimentary phosphates by the hydrochloric acid of the gastric juice. Thus formed, the diffusible glycero-phosphoric acid and soap pass readily into the general circulation, while the emulsified fats find their way through the lacteals and the thoracic duct into the venous current of the blood. Reaching the tissues, the glycerin and fatty acids are readily oxidized, while the stable emulsified fats are deposited in the adipose tissue for a more gradual disintegration.

Besides the fat that is thus received directly with the food, a certain amount of fatty matter is produced during the retrograde metamorphosis of the nitrogenous or albuminoid constituents of the tissues. During this process a portion of the albuminous protoplasm is in part transformed into nitrogenous substances on their way to the formation of urea, while another portion containing no nitrogen is transformed into matter that is identical with fat and the carbohydrates. It thus becomes possible for an animal to grow fat though fed entirely upon albuminoid substances.

If fat be introduced into the blood beyond the capacity of the soaps and other alkaline salts to hold it in solution, the surplus remains suspended in the form of minute oil-globules, giving to the circulatory fluid an oily appearance. This excess constitutes what is termed lipæmia. Under such circumstances, at the ordinary rate of oxidation within the tissues the surplus fat cannot be oxidized, and it accumulates in the cells of the liver and in the adipose tissue. When, however, the process of oxidation is accelerated by exercise or by a febrile condition of the body, the storage of fat ceases and previous deposits rapidly disappear. Under such circumstances the staying power of the individual is notably diminished, since the reserve forces that have been stored up in the adipose tissue are largely exhausted. Hence the lack of endurance that characterizes athletes, boatmen, and pugilists who have been over-trained to a degree that has deprived them of adipose tissue.

When the supply of fat in the blood is too great to be disposed of by immediate oxidation or by deposit in the adipose tissue, it must find its way out of the body. The paths of elimination are through the intestines and the skin. Occasionally it is found in the urine, constituting one of the varieties of chyluria. In the fæces it may be discovered, especially when the pancreatic and biliary secretions are insufficient. The sebaceous glands of the skin excrete excessively, giving to the hair and to the epidermis a greasy and disagreeable appearance. Irritated by this exaggeration of function, the sebaceous glands become inflamed, causing the appearance of pustules of acne and the evolution of eczema. Such eruptions are therefore not uncommon among anæmic patients and among the victims of dyspepsia, irregular menstruation, and nervous disturbances of a depressing character.

Etiology.—Among the causes of obesity Bouchard has noted excess in eating and drinking in about 40 per cent. of the cases; in about 37 per cent.

there was a deficiency of exercise; but in 20 per cent. the amount of exercise was extraordinary; and in 10 per cent. the quantity of food was less than the normal amount.

The disease occurs more than twice as frequently among women as among men, probably on account of the greater activity of the male sex.

Hereditary influences play a very important part in the evolution of obesity. In nearly one-half of the subjects corpulence had been observed among their ancestors. Hereditary influences that predispose to the occurrence of arthritic disease are usually favorable to the development of obesity—a fact that is due to the retardation of nutrition that is a common characteristic of the victims of rheumatism, gout, gravel, biliary lithiasis, diabetes, asthma, acid dyspepsia, hæmorrhoids, hemicrania, neuralgia, and eczema. Through many successive generations these diseases may be traced, either associated at the same time in the same subject or occurring in alternation with each other. A close relationship between this arthritic predisposition and those forms of scrofula that do not develop tuberculosis has often been observed, inasmuch as scrofulous children not unfrequently exhibit arthritic diseases later in life.

The manifestation of obesity may occur at any time during the course of life. It is sometimes a congenital disease; more frequently it begins in childhood; in other cases it does not appear before the age of puberty; and sometimes it is delayed until the declining years of life have been reached.

More frequently obesity is occasioned by an excessive use of starchy and saccharine food than by the consumption of fat. In many cases it is conditioned by the existence of acid dyspepsia, since the action of the pancreatic juice is greatly impeded by excessive acidity in the small intestine: under such circumstances the fats are absorbed in the form of an emulsion instead of being split up into glycerin and fatty acids. Emulsified fats are not easily oxidized, but are stored up in the adipose tissues, where their permanent sojourn is favored by sedentary habits and by insufficient exercise. The daily use of alcohol, even in moderate quantity, interferes with the processes of oxidation, and in like manner favors the accumulation of fat in the adipose tissue. Some of the finest examples of obesity are furnished by individuals who lead a life in the open air without much active muscular exercise. Their appetites are excellent, they eat large quantities of food, and by the use of alcoholic stimulants they favor its permanent storage in the body. Policemen, sea-captains, cavalry officers, and coachmen frequently afford conspicuous examples of the unfortunate consequences of such a life. Very deleterious under such circumstances is the use of malt liquors, since these drinks, besides alcohol, contain a considerable quantity of dextrin and sugar, which aid in the accumulation of fat.

The development of anæmia and obesity is favored by small, but frequent losses of blood. For this reason these diseases are frequently associated in the female sex. The diminished current of blood cannot transport a sufficient amount of oxygen: the exchanges upon which nutrition is dependent are consequently retarded and obesity is developed. In like manner, the disease frequently accompanies pregnancy, excessive menstruation, and lactation, especially if the patient leads a luxurious life and gratifies an appetite for alcoholic beverages and sweet articles of diet.

The period of convalescence after various infective diseases is sometimes characterized by the appearance of a tendency to obesity that is probably due to the existence of conditions that have been already described. Sometimes, undoubtedly, the disease may be directly occasioned by the retardation of nutrition through the agency of the toxic secretions yielded by infective microphytes.

An insufficient oxidation of fat in the tissues has been made prominent as one of the causes of obesity. Not infrequently, however, the disease is determined by the excessive production of fat in the tissues, either as a consequence of an excess of oily substances in the food or as a consequence of the disintegration of albuminoid molecules. Physiological experiment has demonstrated that fat can be accumulated from a diet that is composed entirely of lean meat. A diet that consists chiefly of starch and of sugar favors the deposit of fat, provided a certain amount of albuminoid food be also supplied to the tissues. The carbohydrates under such circumstances are oxidized, while the albuminoids are split up into fat and into simpler nitrogenous compounds, by which the integrity of the tissues is maintained and the adipose tissue is increased. Alimentary substances like potatoes, bread, and meal are therefore favorable to the accumulation of fat, since they contain not only starch, but albuminoid constituents.

Many opinions have been expressed regarding the influence of water upon the development of fat. It has been stated that a copious use of water does not increase the adipose tissue of the body, but in the opinion of others the bodily weight and the adipose tissue are considerably increased by an abundant supply of water. Pure water probably exerts very little influence upon the growth of fat, but when diluted with alcohol and reinforced with sugar and dextrin it undoubtedly favors the development of corpulence.

Obesity is not unfrequently associated with disorders of the nervous system. It is not uncommon among hysterical patients and among the victims of neuralgia. The corpulence that sometimes follows a stroke of paralysis is probably due to the sedentary life that is necessitated by the difficulty of locomotion.

Pathological Anatomy.—As a consequence of obesity, adipose tissue is developed chiefly upon the anterior and lateral portions of the abdomen, upon the loins, hips, breasts, neck, cheeks, and under the chin. It accumulates in the flexures of the joints, upon the palms of the hands, and on the soles of the feet; it grows beneath the aponeuroses, between the muscles, and in the spaces that separate their constituent fibres. Internal organs like the kidneys and the pancreas may be completely buried in masses of fat. The stomach and the intestines are usually dilated; the liver is enlarged, its borders lose their sharpness, its color is pale, and oil oozes from an incised surface; the hepatic cells are gorged with fat, but they are not in a condition of fatty degeneration, and the connective tissue by which they are surrounded is not invaded by fatty particles. The secretion of bile is greatly diminished, and the gall-bladder and larger ducts are often empty or contain nothing but mucus.

When obesity is complicated by chronic alcoholism or by other diseases of the liver, fatty degeneration invades the hepatic cells. The connective tissue then exhibits a certain degree of sclerosis. The substance of the liver is less soft and unctuous, and the sclerotic process in the connective tissue can be traced between the degenerated parenchymatous cells.

When death has been preceded by cardiac failure the hepatic veins are filled with blood, and the liver presents the characteristic appearances that are caused by stagnation of the blood-current.

In many instances the kidneys present no changes worthy of note, but if death has been preceded by a condition of asphyxia the renal veins share in the general distension of the venous system. In severe cases, especially when complicated with alcoholism, the epithelium of the uriniferous tubules may be infiltrated with fat.

The connective tissue of the pancreas is somewhat infiltrated with fat, but the spleen exhibits very little change.

In female patients the uterus and its appendages are firmly packed into the pelvis and surrounded by layers of adipose tissue. The mammary glands are in like manner compressed and atrophied.

The diaphragm is forced upward by the immense masses of adipose tissue that crowd the abdominal cavity. The thoracic cavities are also diminished by the presence of fat in the mediastinal spaces and under the pericardium and pleuræ: a proper expansion of the lungs is thus prevented and a condition of emphysema may be developed.

The right ventricle of the heart exhibits more fat than the left. The entire organ is burdened with adipose tissue; its muscular structure is anæmic, pale, and soft; sometimes all the cavities are dilated, but in certain cases the left ventricle exhibits hypertrophy, while the right heart is dilated. Occasionally atrophy involves the whole heart. As the disease progresses the muscular fibres are crowded asunder by the invasion of fat, and finally the muscular tissue itself experiences fatty degeneration.

As a consequence of the presence of minute particles of undissolved fat the amount of oil in the blood may be four or five times greater than it should be, constituting 5 or 6 parts per 1000 of the circulating fluid.

Symptoms.—Among adults of medium height and symmetrical proportions a weight of not less than 200 pounds nor exceeding 220 pounds indicates the first degree of obesity. If the weight reach 300 or 350 pounds, the patient exhibits the medium form of obesity. A weight between 400 and 500 pounds indicates a very grave form of the disease. Still greater weights have been recorded, and there is related the case of a man who measured fifteen feet around the waist and weighed 715 pounds; but this weight is quite moderate in comparison with that of another patient, who is said to have weighed 1070 pounds.

The adipose tissue in cases of obesity is not always distributed uniformly throughout the body. Its special accumulation in certain organs and regions depends largely upon the sex and the habits of life of the patient. Women often exhibit an excess of fat about the shoulders and breasts. If the habits of life be sedentary and accompanied by the habitual gratification of an excellent appetite, the abdominal walls exhibit an enormous prominence. The disease may reach such a degree that locomotion becomes exceedingly laborious. The various movements that are needful for the accomplishment of the different acts of life are rendered impossible without assistance, and it becomes finally necessary for the victim of obesity to remain continually in bed, supported in a sitting posture with numerous pillows and cushions about the person, since it is impossible to lie down without a sensation of suffocation.

Under the influence of obesity the sexual appetite rapidly fails and is prematurely extinguished. Among young girls who exhibit the disease menstruation usually appears at a precocious age: it is generally irregular, and alternating conditions of amenorrhœa and menorrhagia are not uncommon. In the married state sterility is a frequent consequence of the disease.

As a rule, the strength of the muscular apparatus is greatly diminished, though in certain instances a great degree of skill and dexterity in the use of certain muscles has been cultivated by professional musicians, artists, and gymnasts who were victims of obesity. Occasionally a respectable degree of intellectual ability is exhibited by very corpulent people, but, as a general rule, the functions of the brain are greatly retarded, so that the term "fat-witted" has passed into a proverb. Much of the time of such persons is passed in sleep, and their meals are ordinarily followed by a period of profound somnolence.

The movements of respiration and the action of the heart are greatly impeded by the existence of obesity. The limited capacity of the thorax renders it impossible to breathe deeply and sufficiently during unusual muscular exertion; consequently, the patient is soon out of breath after the slightest effort. The amount of hæmoglobin in the red blood-corpuscles is also diminished in many instances, though the number of the corpuscles does not fall below the normal figure. The blood cannot take up and transport a sufficient supply of oxygen under such conditions, and consequently a sense of breathlessness is often experienced.

The pulse is retarded in about one-third of the cases; in another third it is feeble, frequent, and dicrotic. The heart itself exhibits signs of weakness, and palpitation accompanies the dyspnœa that arises in connection with muscular exertion. Among elderly subjects the symptoms of arterio-sclerosis appear in about one-quarter of the cases. The pulse is full and strong; vertigo, dyspnœa, and asthma frequently occur. Occasionally there is intense dyspnœa, accompanied by a feeble, frequent, and irregular pulse, indicating great cardiac debility.

Hæmorrhagic effusions, taking the form of epistaxis, hæmoptysis, and bleeding piles, are frequently observed among the obese. Such hæmorrhages are principally conditioned by fatty degeneration of the vascular walls.

Two forms of obesity have been remarked, and they are easily distinguished from each other. The first is characterized by a condition of plethora: the face is flushed, the lips are blue, and the superficial veins upon the cheeks and nose are distended with blood. The second or anæmic form of obesity is characterized by pallor of the skin, by the existence of murmurs in the large vessels, and by a tendency to fainting on slight provocation. In certain chlorotic cases accompanied by obesity the vascular organs are congenitally deficient; the heart, lungs, and liver are disproportionately small, and the diminutive arteries are insufficient for the distribution of blood throughout the relatively enormous body. In such cases, though the quality of the blood may not be deficient, its quantity is less than the normal amount.

In the protoplasmic elements of the tissues the process of oxidation is retarded; consequently, the amount of carbonic acid that is formed and discharged through the lungs exhibits considerable diminution, and the temperature of the body becomes subnormal. In the majority of cases the amount of urea falls below the proper figure, though it sometimes remains normal in quantity, and occasionally may be actually increased. The discharge of phosphates is usually diminished.

When an excessive accumulation of fat is caused by the degradation and splitting up of the albuminous constituents of protoplasm, a portion of the imperfectly oxidized nitrogenous remainder appears in the urine in the form of a modified albumin that is not dependent upon inflammation in the kidneys, but is the representative of the disorder that has overtaken the processes of nutrition. Such albumin is non-retractile, is diffused throughout the urine, and is indicative of cachexia rather than of actual renal disease. Sometimes the urine contains minute oil-globules, and the presence of sugar may be frequently discovered. The occurrence of glycosuria is often due to the coexistence of diabetes with obesity, but it is sometimes dependent upon a disorder of the liver that interferes with the transformation of alimentary starch and sugar into glycogen. In such cases glucose passes directly from the portal vein into the general circulation, and is eliminated with the urine.

The urine in cases of obesity very often contains oxalate of calcium, because the retardation of oxidation in the tissues interferes with the oxidation and destruction of the oxalic acid that reaches or is formed within the tissues. For a similar reason the volatile fatty acids escape oxidation, and they are eliminated in considerable quantity through the glands of the skin, where they cause irritation and inflammation of the sebaceous follicles and of the integument. For this reason obese patients frequently suffer with acne, eczema, intertrigo, and seborrhœa. The copious perspiration frequently exhales a disagreeable odor that is due to the presence of imperfectly oxidized volatile substances.

In about 50 per cent. of the cases of obesity the appetite is normal; in about 10 per cent. it is reduced, and the daily consumption of food is less than usual in health. In a considerable number of cases there is great hunger, and the patient devours an enormous quantity of food. Thirst is sometimes excessive, and is significant of the existence of polyuria. In the majority of patients the power of digestion gradually fails; the stomach becomes dilated; and the symptoms of acid dyspepsia (pyrosis, flatulence, and constipation) are developed. As a consequence of chronic catarrhal enteritis obstinate diarrhœa may afflict the patient, and large quantities of undigested fat may be discovered in the fæces.

In many instances obesity is complicated by stagnation of the blood in the intestinal vessels. The muscular layer of the intestinal wall becomes enfeebled, there is obstinate constipation, and hæmorrhoids may result from dilatation of the rectal veins. In like manner, the veins in the lower limbs and scrotum become dilated and varicose. A similar loss of contractility in the non-striated muscular tissues of the body is universally manifested in a large proportion of the cases of obesity.

Treatment.—From time immemorial the importance of moderate diet and abundant exercise has been recognized in the treatment of obesity. The exclusion of fat and starches from the food, together with great moderation in the matter of liquid nourishment, will accomplish rapid diminution in the weight of the body; but under such a restricted diet a tendency to lithiasis is liable to be developed, and then the patient may suffer from hepatic or renal colic, caused by the presence of gall-stones or renal calculi. It is for this reason that the well-known Banting method for the reduction of corpulence is often a cause of ill-health that is worse than the original disease. Ebstein recommends three meals a day, limiting the breakfast to one cup of black tea, without milk or sugar, with two ounces of well-buttered toast. Dinner should be taken in the middle of the day, and it may consist of shin-bone soup, followed by four to six ounces of well-boiled or roasted fat meat served with gravy; peas, beans, and cabbage may be allowed in moderation; the dessert should consists of salads, fresh fruit or dried fruits with sugar, and a moderate quantity of light wine and black tea without milk or sugar. The last meal of the day should consist of a single cup of black tea without milk or sugar, an egg or a little fish, ham, or other fat meat instead, an ounce of well-buttered bread, and a little cheese or fresh fruit. A certain amount of fat must be supplied with the food, but starches and sugars are to be avoided. Oertel and others unite in recommending a similar dietary.

Dujardin-Beaumetz has shown that such a restricted diet is insufficient for the maintenance of healthy nutrition. If the different organs of the body have not yet undergone degeneration, he allows the patient to indulge in about half a pint of liquid with each meal. If wine is drunk, it should be very light and must be diluted with Vichy or other alkaline waters. For this purpose the Londonderry lithia water may be recommended. If, however, it should become necessary to avoid too great dilution of the gastric juice, the patient should abstain from drinking at meal-time, and may be directed to drink a pint of weak tea without sugar about two hours after each meal. Beer and strong alcoholic beverages must be avoided. A little black coffee may be sometimes permitted at the conclusion of breakfast. Soup must not be taken, but eggs, 'fish, meat, fresh vegetables, and fruits, like oranges, that are not too starchy, are permitted. Pastry is forbidden. The crust of bread is preferable to the crumb. Vienna rolls and soup-sticks, which consist largely of crust, are to be preferred. Breakfast should consist of such bread to the amount of three-quarters of an ounce, together with an ounce and a half of cold meat and a cup of weak tea without sugar. For luncheon the patient should take an ounce and a half of bread, three ounces of meat or two eggs, the same quantity of fresh vegetables, salad, half an ounce of cheese, and fruit to suit the appetite. Dinner should be served in the evening without soup, and in addition to the articles prescribed for luncheon the patient may take an ounce and three-quarters of bread and three ounces of meat.

Bouchard bases the treatment of obesity upon the condition of the urine. When nitrogenous excreta are present in excessive quantity the amount of albuminous food should be reduced. When urea is deficient in the urine the quantity of food should be at first diminished, and then increased as convalescence progresses. In all cases the quantity of fat, starch, and sugar in the food should be five times greater than the amount of nitrogenous nutriment. The quantity of fat and of sugar should be somewhat reduced, so as to avoid any unoxidized surplus, but the processes of oxidation may be accelerated by increasing the ingestion of vegetable acids. This may be accomplished by eating fruits and fresh vegetables that contain organic salts of potassium. Vinegar and other free acids should be avoided, since their considerable use causes emaciation through the destruction of the healthy framework of the cells. The use of acids in small quantities aggravates obesity by diminishing the alkalinity of the blood ; but when combined with potassium the resulting salts favor the oxidation and destruction of fat. For a similar reason food should not be taken too frequently or in too large a quantity, and the patient should take active muscular exercise before meals. If, however, there be an excessive elimination of urea and the phosphates, the processes of oxidation should not be thus unduly stimulated.

In order to promote the processes of nutrition it is desirable that the trophic functions of the nervous system be aroused by agreeable occupation, travel, and moderate intellectual excitement. A favorable result is often obtained from massage, hydropathy, and other methods by which the skin is stimulated. The function of the liver must be regulated in order to promote intestinal digestion and to avoid constipation. These processes may be influenced in a salutary manner by the use of Carlsbad and other mineral waters which contain the carbonate and sulphate of sodium. The oxidation of fat is also assisted by the use of sodium bicarbonate, liquor potassæ, and small quantities of soap taken in the form of a pill. Warm baths at a temperature of 100° F., continued for half an hour or longer, serve to raise the temperature of the body, accelerate the absorption of oxygen, and promote the discharge of carbonic acid.

In the majority of cases the treatment should be inaugurated by reducing the quantity of food, maintaining the proper ratio between its nitrogenous and its carbonaceous constituents. For this purpose milk and eggs furnish the best nutriment, according to the method that has been popularized by Weir Mitchell and his pupils. The patient should take for twenty days in succession not more than half a pint of milk and one egg every three hours when not asleep. Other food and drink should be forbidden. Thus nourished, the patient becomes constipated, and the bowels require gentle laxatives or enemata. It is not an uncommon experience to suffer at first from a sensation of faintness, weakness, or even dizziness, but in a short time these disagreeable symptoms disappear and superfluous corpulence rapidly diminishes. At the end of three weeks the dietary may be gradually varied and enlarged in accordance with the rules that have been formulated by Dujardin-Beaumetz and other physiologists.

BILIARY LITHIASIS.

By HENRY M. LYMAN.

Biliary calculi are chiefly formed by the precipitation of cholesterin from the bile. This highly carbonaceous substance exists in the bile, blood, and nervous tissues. As an excrementitious substance it is found in the fæcal contents of the intestine. It is not certain whether it is derived principally from the tissues or whether it is transported by the red corpuscles to the tissues. Austin Flint, however, has shown that cholesterin is more abundant in the blood of the cerebral veins than in the contents of the cerebral arteries —a fact that favors the hypothesis of its origin in the retrograde metamorphosis of the cerebral tissues.

The precipitation of cholesterin does not take place in the tissues, because it is held in solution by lecithin. In the circulating fluids of the body its solubility is maintained by the alkaline salts and by the compounds of potassium and sodium with the fatty acids. In the bile, so long as that liquid is alkaline, cholesterin is held in solution by the biliary salts.

The introduction of calcium into a liquid that contains cholesterin causes a precipitation of that substance, since calcium combines with the fatty acids and biliary acids to form insoluble salts which are powerless to maintain cholesterin in a state of solution. Any excess of organic acids in the liquids and solids of the body causes a liberation of calcium from the anatomical elements in which it exists, and this liberation of basic calcium is speedily followed by the precipitation of cholesterin.

The excessive presence of cholesterin in the fluids of the body is liable to be followed by its precipitation, an event that is very apt to occur when the bile stagnates and becomes concentrated in the biliary passages.

The formation of biliary calculi is usually determined by the presence of particles of solid matter accidentally present in the biliary passages. Intestinal parasites and their ova that have found their way into the biliary passages may become centres of crystallization, around which considerable masses of cholesterin are accumulated, producing gall-stones or biliary calculi, which originate intense pain as they pass through the hepatic or cystic duct.

Etiology and Pathological Anatomy.—Biliary calculi are very frequently discovered in old people. The female sex is more liable than the male to experience the disease, partly on account of a greater tendency to sedentary habits, and partly by reason of the predominance among females of osteomalacia, a disease that favors the liberation of calcium bases from their combinations in the bony skeleton.

The occurrence of biliary lithiasis is also favored by excessive eating and drinking, especially when associated with an insufficient amount of exercise. Intellectual and emotional disturbances of the nervous system which interfere with nutrition are often favorable to lithiasis; in short, everything that retards the processes of oxidation exerts a similar influence.

The association of a tendency to biliary lithiasis with those disorders which characterize the arthritic diathesis is a matter of common observation. Various forms of eczema, erythema, urticaria, coryza, tonsillitis, recurring attacks of bronchitis, neuralgia, hemicrania, lumbago, congestive headache, hæmorrhoids, chronic dyspepsia, gravel, rheumatism, gout, asthma, obesity, and diabetes are most intimately connected with the occurrence of biliary calculi.

The number and size of the concretions that may be formed in the biliary passages is exceedingly variable. As many as seven thousand gall-stones have been discovered in a single individual. Sometimes the gall-bladder is occupied by a single stone, but usually the number is more considerable. The calculi are moulded upon one another, presenting numerous facets caused by mutual pressure. Occasionally external pressure upon the abdominal wall causes a grating sound that is actually audible at a distance from the body. The centre of the calculus usually consists of a pigmented mass of calcium combined with biliary or fatty acids. Upon this the crystals of cholesterin are deposited, sometimes in alternation with layers of calcareous salts. In certain cases the nucleus of the concretion is composed of epithelium that has been thrown off from the lining of the biliary passages under the irritating influence of concentrated bile, or as a consequence of the entrance of intestinal microphytes which have found their way into the biliary passages. Occasionally gall-stones consist entirely of pigment or of carbonate of calcium, but the mixed calculi are far more numerous.

The presence of gall-stones in the bladder frequently excites the inflammation of its internal surface. The muscular coat of the cyst may undergo fatty degeneration, calcification, and atrophy. Sometimes suppuration takes place, and the gall-bladder becomes transformed into an abscess that may open into the peritoneal cavity, into the stomach or intestines, or may penetrate the abdominal wall.

Similar changes involve the hepatic biliary passages when calculi are formed within those ducts.

Symptoms.—The characteristic symptom of biliary lithiasis is the occurrence of severe pain of a paroxysmal character in the region of the gall-bladder, unattended by fever, lasting sometimes for a number of hours or for two or three days, and followed by a moderate degree of jaundice. Frequent paroxysms of this sort should arouse suspicion of the existence of biliary calculi; and this will be rendered still more probable if an oval tumor can be detected in the situation of the gall-bladder. Sometimes, when the abdominal walls are unusually thin, the smooth and rounded outlines of the cyst can be distinctly felt, and occasionally the calculi can be distinguished, or even be made to move upon one another with an audible sound when pressed with the fingers. In certain cases, however, no symptoms of their presence are observed until the occurrence of an abscess in the abdominal wall opening externally and giving exit to biliary sand and calculi, along with bile and mucus, reveals the fact of chronic lithiasis.

If the formation of calculi principally involves the hepatic ducts, pain and jaundice are the prominent symptoms, unless suppuration should lead to the formation of an abscess in the substance of the liver around the impacted calculus. Careful scrutiny of the fæces after a paroxysm of hepatic colic sometimes reveals the presence of calculi that have escaped from the common bile-duct and have entered the intestinal canal. After every such paroxysm the stools should be received in a vessel and be thoroughly mixed with water, which should be then passed through a sieve for the purpose of retaining all insoluble matter. In this way gall-stones may be sometimes discovered.

In certain cases, despite repeated paroxysms of pain, no discharge of calculi into the intestine can be effected. Inflammation, ulceration, and perforation of the gall-bladder or of the biliary ducts may then occur, leading to an escape of calculi into the peritoneal cavity. Circumscribed peritonitis sometimes forms adhesions by which the intruded masses are confined, thus protecting the remainder of the peritoneal sac. Not infrequently, however, a second rupture occurs, and the contents of the inflammatory cyst are diffused throughout the peritoneal cavity, producing general peritonitis and death.

Diagnosis.—The symptomatic pain of biliary calculi must be distinguished from gastralgia by the situation of the pain, by the absence of gastric disorders, and by the presence of icterus.

Ordinary intestinal colic is characterized by the flatulent condition of the intestines and by an increase of pain on pressure.

When the colic is due to lead-poisoning the bowels are usually constipated and other symptoms of plumbism are present.

Hepatic neuralgia is characterized by pain in the vicinity of the liver, accompanied by evident neuralgia in other parts of the body.

Typhlitis, perityphlitis, and paratyphlitis are characterized by the existence of a painful inflammatory swelling in the right iliac fossa.

Renal colic is characterized by the presence of blood and renal sand or calculi in the urine. The pain also follows a line from the kidney along the ureter and the bladder and testicle.

The pain and tenderness that are caused by a floating kidney might be sometimes mistaken for biliary colic, but the form and situation of the tumor upon the right side considerably below the liver should correct such an error.

The pain of intestinal obstruction might be, under certain circumstances, mistaken for biliary colic, but the diagnosis will be enlightened by the occurrence of obstinate constipation and vomiting, the latter being oftentimes fæcal, in obstruction of the bowel.

It is scarcely possible that a psoas abscess should be confounded with the existence of biliary calculi, but it is said that the presence of a large gall-stone in the intestinal canal has been actually mistaken for that disease.

Aneurismal tumors within the abdomen may be distinguished from biliary accumulations by the characteristic pulsation of an aneurism. In certain cases, marked by paroxysmal pain, chilliness, and fever, the existence of malarial poisoning may be suspected; but the course of the disease and the inefficacy of quinine will serve to distinguish the biliary disorder.

In like manner, the vomiting, retching, and cramps that accompany cholera morbus might be occasionally confused with an attack of biliary colic.

Finally, a sudden, rapidly progressive, and fatal attack of biliary colic might easily suggest the idea of some form of poisoning.

Treatment.—The medicinal treatment of biliary lithiasis consists in the management of the paroxysm of colic and in the administration of remedies for the removal of calculi. During a paroxysm of pain hypodermic injections of morphine with large doses of chloral hydrate must be administered. A warm poultice should be placed over the liver, and the patient may be allowed to drink alkaline mineral waters or water containing a small quantity of sodium bicarbonate. During the interval between successive paroxysms the patient should take eight or ten grains of salicylic acid or of salicylate of sodium three times a day for a long period of time. Alcoholic stimulants should be avoided, and robust individuals may drink Carlsbad water or other strong alkaline laxative mineral waters. Sodium phosphate may be prescribed in drachm doses, two or three times a day, with considerable advantage. Each dose should be taken with copious draughts of hot water before meals. Chloroform has been recommended in doses of ten drops every three hours; it may be suspended in milk. Ether and the oil of turpentine are frequently employed with the hope of preventing the formation of calculi: five drops of turpentine, with twenty drops of ether, may be given four times a day. Large doses of olive oil have been frequently administered for the purpose of evacuating gall-stones, and their use has sometimes proved satisfactory, so far as the relief of pain has been concerned, but it has been clearly shown that the inspissated masses that are voided from the intestines after such medication consist of transformed mucus and oleate compounds that have been formed in the intestinal canal.

The physiological treatment of biliary lithiasis consists in modification of the diet, by which the formation of cholesterin is diminished, the bile is diluted, and the conditions that favor the precipitation of cholesterin are annulled. For this purpose animal food should be restricted in amount, since it contains a considerable quantity of cholesterin. The patient should be encouraged to drink large quantities of water in order to dilute the bile. If the condition of the stomach be such as to render large draughts of water objectionable, a favorable result may be obtained by frequent irrigation of the bowels with copious rectal injections. Stagnation of the bile in the biliary passages must be prevented by the administration of cholagogue laxatives, like rhubarb, aloes, podophyllin, and euonymin. It is not necessary to prohibit the use of fats, since a certain amount of such substances is needed for healthy nutrition. Fresh vegetables and fruits are useful on account of the organic acids and alkaline salts that they contain. The organic salts of potassium are especially useful, because by their presence they prevent the liberation of calcium, the substance which favors the precipitation of cholesterin. Mineral waters containing carbonic acid and calcium salts should be forbidden. Champagne and other artificial drinks that are rich in carbonic acid are also to be avoided. Pure rain-water and distilled water that has been aërated are the safest of all beverages. Sugar and starch should be used in moderation, since they appropriate much of the oxygen that is needed for the proper oxidation of the organic acids in the tissues. Red wine may be allowed when gastric catarrh does not exist, and coffee may be drank if the condition of the nervous system does not contraindicate its use.

GRAVEL.

BY HENRY M. LYMAN.

Definition.—The term "gravel" is employed to indicate those concretions of a mineral or organic character which are formed in the excretory passages of the kidneys. When formed within the bladder itself such concretions are distinguished by the term "vesical gravel." The masses which are thus formed are exceedingly variable in size, sometimes appearing as an almost impalpable powder, and again forming genuine calculi that are voided with difficulty and pain. In fact, the line of demarcation between gravel and calculus is very indefinite so far as the size of individual particles is concerned. It is convenient, however, to restrict the term "gravel" so that it shall only include such concretions as can be readily expelled through the ureters and urethra.

Etiology.—Three principal forms of gravel are encountered. Of these the phosphatic variety is dependent upon local causes, usually of an inflammatory character. As a consequence of catarrhal inflammation or ulceration of the mucous membrane of the bladder a deposit of phosphatic concretions takes place. These incrustations are formed principally of the phosphate and carbonate of calcium. The urine under such circumstances finally becomes ammoniacal, and masses of ammonio-magnesium phosphate are then formed. This ammonio-magnesium phosphate results from fermentation of the urine within the bladder under the influence of the bacteria of fermentation and putrefaction which find their way into the vesical cavity when proper cleanliness is not observed in the use of catheters, sounds, and other instruments that are introduced into the bladder.

The two other forms of gravel are those due to uric acid and oxalic acid; their formation is conditioned by disturbances in the processes of nutrition.

Gravel occurs more frequently among the inhabitants of temperate climates than among the natives of either the polar or the equatorial regions of the earth, probably because it is in the temperate zone that wealth and luxury are most frequently enjoyed. Sailors and nomads who live continually in the open air hardly ever experience the disease, probably for the same reason that gravel, while unknown among wild animals in a state of nature, is very common among similar animals caged in menageries.

The disease occurs among men more frequently than among women, in the proportion of five to one. It is a disease of adult life; children and old people suffer more frequently from vesical calculi. Infants and young people who are badly fed not infrequently suffer from the existence of calculous masses composed of oxalate of calcium, but among well-fed and luxurious adults the occurrence of nephritic colic is usually caused by the formation of concretions of uric acid.

Gravel is usually encountered in association with the other maladies to which the arthritic diathesis is related—viz. eczema, impetigo, erythema, urticaria, coryza, arthritic bronchitis, neuralgia, hemicrania, congestive headache, lumbago, hæmorrhoids, acid dyspepsia, rheumatism, gout, asthma, obesity, diabetes, and polyuria.

The most common form of gravel is the uric-acid variety: it is dependent upon a disturbance of the normal processes of retrograde metamorphosis in the nitrogenous tissues of the body. As a consequence of retarded oxidation the amount of uric acid is abnormally increased and its solubility is also diminished.

Without undertaking to discuss the precise mode of formation of uric acid, it is sufficient to remember that it is a body which is not readily soluble in water. In the urine it is held in solution by the presence of tribasic phosphates, which surrender one equivalent of base for the formation of urates that remain in solution. Now, although under the influence of faulty alimentation and insufficient exercise the amount of uric acid may be greatly increased in the tissues, its compounds will still remain soluble in the urine, provided the conditions for the formation of soluble urates are not annulled. If, however, the urine becomes highly concentrated through insufficient supply of water or by reason of excessive perspiration, or if rendered unusually acid by the presence of acid phosphates instead of tribasic phosphates, the precipitation of uric acid will occur. Everything that favors retardation of the nutritive processes, therefore, tends to facilitate the deposit of uric acid within the urinary passages.

Symptoms.—The existence of gravel is indicated by its appearance as a sediment in the urine, and by the occurrence of uneasiness and pain in the loins, relieved by the free discharge of such sediment. Only when the particles reach a considerable size is the characteristic pain of renal colic experienced. Pain is then felt in the line of the ureter, extending into the bladder and along the spermatic cord. The testicle is frequently retracted. The urine is dark colored, and is sometimes mixed with blood derived from the lacerated walls of the excretory ducts of the kidney. Pain occurs intermittently, and is excruciating if the calculus become incarcerated in the ureter. It is not accompanied by fever unless inflammation and ulceration are excited.

Treatment.—Since the formation of uric-acid gravel is dependent in part upon an increased production of uric acid, and in part upon its precipitation within the uriniferous ducts, it is important that its production should be hindered and that its solubility should be maintained. In order to hinder the formation of uric acid, and in order that there may be less nitrogenous waste that can be transformed into uric acid, the nitrogenous constituents of the food should be reduced in amount, and the formation of urea and hippuric acid, which are soluble, should be promoted. Since the amount of urea cannot be increased by modifying the diet without increasing the amount of alimentary nitrogen, it is necessary to take measures for increasing the formation of hippuric acid. This can be done by furnishing benzoic acid or quinic acid for combination with glycocoll. These acids exist in the external cuticle of fresh vegetables and fruits. For this reason the daily use of unpared apples has an excellent effect as a means of obviating a tendency to uric-acid gravel. The potassium salts that exist in such fruits and vegetables favorably influence the alkalinity of the blood, and thus hinder the precipitation of uric acid. The calcium salts that are present in such fruits and vegetables are only injurious in case of a gravel that consists of the carbonate and phosphate of calcium or of oxalic acid.

The ingestion of sugar and starch, alkalies, and beverages that are charged with carbonic acid should be restricted, since they favor the production and precipitation of uric acid.

Concentration of the urine should be prevented by copious draughts of water. Hot water drunk freely at bed-time has an excellent effect upon the general nutrition of the body. Alkaline waters and distilled waters exercise a similar influence. For this reason copious libations of Londonderry lithia water, Vichy water, Waukesha water, and other mild alkaline waters are very beneficial in the treatment of uric-acid gravel. Recently piperazine in doses of fifteen to thirty grains has been highly praised as a solvent of uric acid.

SACCHARINE DIABETES.

BY HENRY M. LYMAN.

Definition.—Diabetes is a constitutional disorder of nutrition accompanied by persistent glycosuria, and usually characterized by an excessive discharge of urine, inordinate hunger and thirst, progressive exhaustion, and a fatal termination. Accidental and temporary appearance of sugar in the urine does not constitute diabetes, but is termed *glycosuria.*

SYNONYMS.—Diabetes mellitus, Mellituria, Glycosuria, Glucosuria, Diabéte sucrè, Zuckerharnruhr.

Etiology.—While it is true that differences of climate exert very little influence over the occurrence of diabetes, the disease is encountered more frequently in certain localities than in others, for the reason that the character of the food and the mode of life among certain races are especially favorable to the development of arthritic disorders. It is not true that the excessive use of saccharine and starchy food is alone a cause of diabetes; but it is a fact that habitual dietetic errors may occasion a predisposition to the disease through derangement of the processes of nutrition. This is especially true when the cause consists in the inordinate consumption of animal food, together with beer and wine, which, besides alcohol, contain considerable quantities of dextrin and glucose. A similar predisposition is also nurtured by sedentary habits and by neglect of muscular exercise. The influence of an hereditary disposition is as apparent in this connection as it is in other maladies that are dependent upon disorders of nutrition, such as obesity, gout, rheumatism, lithiasis, asthma, certain forms of eczema, hæmorrhoids, etc. Diabetes is also a rather common heritage among the descendants of neurotic families, in which mental derangements, epilepsy, paralysis agitans, exophthalmic goitre, and hysteria have been remarked. The coexistence of obesity and diabetes is a matter of frequent observation. Both sexes are about equally liable to the disease, but it is most common in later life, during the period between fifty and seventy years of age.

The exciting causes of diabetes are frequently connected with the occurrence of acute and chronic diseases of the brain. Tumors and injuries, especially if they involve the fourth ventricle, are often accompanied by the development of diabetes. In many instances the disease has its origin in psychical causes: disappointment, grief, business failures, and all emotions of a depressing character are often followed by a development of diabetes. It sometimes follows the course of the infective diseases, and it may be excited by disorders of the liver, pancreas, and other digestive organs.

Pathological Anatomy.—In the majority of cases of diabetes the lesions that are found in the brain after death are usually consequences rather than causes of the disease. It is not uncommon to find evidences of œdema, congestion, and thickening of the meninges, together with anæmia, atrophy, and softening of the convolutions of the brain, and sclerosis or amyloid degeneration of the cerebral tissues. Cysts have been sometimes discovered in various parts of the cerebral substance, but it is when tumors are developed in the region of the medulla oblongata and the fourth ventricle that the occurrence of glycosuria can be confidently ascribed to the direct influence of such neoplasms. In certain cases the nervous phenomena that have been displayed in connection with the progress of the disease have been attributed to obstruction of the vascular channels by embolic masses of glycogen that have become lodged in the small vessels of the bulb and of the brain. In the spinal cord no characteristic lesions have been observed, but the cranial nerves may be subjected to pressure by tumors which sometimes serve as exciting causes of the disease. Inflammation of the spinal nerves is sometimes observed, and may be the result of an excess of sugar in the blood by which they are nourished. In certain cases there are enlargement and induration of the ganglia of the sympathetic nerves. The general appearance of the blood is usually unchanged; the number of the red blood-corpuscles exhibits no diminution until the later stages of the disease are reached, but the white corpuscles are often notably increased in number. It is the presence of sugar that produces the most characteristic change in the quality of the blood. The amount is, however, quite variable. Urea and other nitrogenous excreta are often present in excessive quantity. Occasionally acetone has been detected.

The heart is pale and soft, and in about 50 per cent. of the cases it is either enlarged or fatty. Pericarditis and endocarditis have sometimes been discovered. The arterial walls often exhibit a condition of fatty degeneration or atheroma. The liver is often enlarged and fatty; cirrhosis frequently exists, and sometimes the organ exhibits pigmentary degeneration. The spleen is usually small and soft, and its arterial structures are in a condition of hyaline degeneration. The pancreas is often found to have undergone atrophy, sometimes as a consequence of sclerosis of the connective tissue, and sometimes as a result of the formation of calculi that obstruct the excretory ducts of the glands; occasionally the obstructed ducts become dilated so as to form cysts. It should be remembered that compression of the pancreatic duct by any new growth or mass of exudation may produce atrophy of the gland, and when the pancreas is thus disorganized a malignant form of diabetes is speedily developed, and results in rapid emaciation and an early death. Glycosuria seldom follows simple obstruction of the pancreatic duct, but it is almost invariably observed when the gland is invaded by a perivascular sclerosis which limits the secretory functions of the organ, and is equivalent to its total extirpation. In the stomach and intestines the mucous membrane is frequently found in a condition of chronic catarrhal inflammation. The lungs and bronchi also are often inflamed, but they are more frequently involved in a process of tubercular infiltration. Renal disease may exist as an accidental complication of diabetes, but in many instances the kidneys are diseased as a direct consequence of the glycosuria. The epithelium of the convoluted tubules and of certain portions of the straight tubules sometimes exhibits a granular degeneration similar to that seen in coagulation-necrosis. Infiltration with glycogen is often discovered, involving the epithelial cells of the uriniferous tubules within the marginal zone.

Chronic inflammation frequently affects the mucous membrane throughout the entire urinary tract. Balanitis and vulvitis are not uncommon consequences of the proliferation of pyogenic microphytes in the saccharine fluids that irrigate the mucous membrane and surfaces in the vicinity of the meatus urinarius.

For a similar reason various cutaneous diseases frequently appear during the course of diabetes. The entire body is soaked with a saccharine fluid that is admirably adapted for the growth of a luxuriant vegetation of parasitic microphytes that irritate the surface of the skin. Among these disorders may be mentioned a species of lichen that closely resembles xanthelasma. Other parasitic diseases, such as boils, carbuncles, phlegmons, and gangrene of the skin and subcutaneous tissues, are of frequent occurrence. Within the eyeball opacity of the lens, producing cataract, is the most common lesion. Retinal hæmorrhages are sometimes witnessed, and the retina or the optic nerve may undergo atrophic degeneration.

Symptoms.—Saccharine diabetes is developed in a manner so insidious that only after a considerable period of time are its symptoms sufficiently marked to excite attention or suspicion regarding the nature of their cause. An examination with a view to life insurance or for some other comparatively trifling purpose often affords the first intimation of the existence of glycosuria. In many instances an excellent appetite is present during the early stages of the unsuspected disease, but gradually there is an apparent loss of vigor ; the patient becomes thirsty ; the tongue grows red and dry ; the flow of urine is increased, and wherever it sprinkles the clothing it leaves, as it dries, a white powdery deposit of sugar. In certain cases there is intolerable itching in the neighborhood of the urethral meatus ; perspiration ceases ; the nails become brittle, and sometimes are shed without any apparent cause ; the surface of the body is irritated by obstinate itching eruptions ; boils and carbuncles and gangrenous patches are developed in the skin ; sexual appetite and vigor disappear ; the eyesight becomes enfeebled ; the body becomes rapidly emaciated, or if the weight be not diminished there is excessive reduction of muscular power. Death finally occurs, either from exhaustion or in a condition of coma or as a result of various complicating diseases.

The invasion of the disease is so insidious, and its evolution is so exceedingly variable, that it is seldom possible to fix the date of its commencement. In the fully-developed stages of diabetes the concurrence of glycosuria, polyuria, excessive hunger and thirst, emaciation or exhaustion, forms a group of pathological symptoms of the most conspicuous and unmistakable character.

The urine in diabetes is pale and opalescent; its odor resembles that of ripe fruit, and when it contains 3 or 4 per cent. of sugar it has a sweetish taste; the specific gravity is frequently and rapidly variable. When the quantity of urine is not considerably increased the specific gravity exceeds the normal figure. The acidity of the liquid is usually excessive, so that the urinary passages are often irritated and inflamed by the acid excretion. In the bladder, however, such accidents are more frequently caused by the process of fermentation.

The mere presence of copper-reducing substances in the urine is not decisive of the existence of diabetes, since saccharine urine is occasionally voided during the course of various diseases; and copper-reduction may occur as a consequence of intoxication with chloroform, nitrous oxide, and other substances. The existence of genuine diabetes can be affirmed only when there is permanent glycosuria, or when the amount of sugar that is discharged exceeds the daily normal production of sugar in the liver (3000 grains). Permanent glycosuria is the most important symptom, since in many cases the amount of sugar is comparatively small. The urine that is voided during the period of digestion is highly charged with sugar, but during the later stages of the disease the largest amount is voided with the morning urine, since that represents the waste of the tissues during sleep, when the processes of disintegration are most active. During intercurrent diseases and during catarrhal disturbances of the alimentary canal the urine may be temporarily free from sugar, but it reappears with the restoration of the ordinary condition of health.

As diabetes progresses the quantity of urea is often increased, though it subsequently diminishes during the period of terminal cachexia when the processes of nutrition and of disintegration are all reduced to a minimum.

The disintegration of the nitrogenous elements of the tissues is accompanied by a corresponding increase of phosphates in the urine: this increase is principally occasioned by the breaking up of the albuminous constituents of the body. The formation and discharge of urea and of the phosphates do not always proceed on parallel lines. Sometimes one or the other substance is in excess, and sometimes there is an alternation of excessive discharge in the case of either class. The phosphaturia that thus occurs is maintained at the expense of the solid elements of the bony skeleton, hence the liability to fracture of the bones that is sometimes observed during the course of diabetes.

Albuminuria is often associated with glycosuria during the course of the disease. In certain special cases and during the later stages it is due to structural changes in the kidneys, but in the majority of cases the amount of albumin is very trifling. Such forms of albuminuria are often of brief duration, and are not accompanied by other typical evidences of renal disease. It is witnessed principally among patients who manifest the symptoms of diabetic phthisis, and it is an evidence of cachexia rather than of specific disorganization.

Uric acid often appears in excessive quantities before the occurrence of glycosuria. It is often discharged in an inordinate quantity during the course of the disease—a fact that marks the close relationship between diabetes and gout.

The polyuria that usually accompanies diabetes is especially exaggerated at night; the total amount of urine that is voided during twenty-four hours varies from two to fifteen quarts, according to the degree of thirst that is experienced and the amount of water that is drunk by the patient. In severe forms of diabetes there is a consuming thirst; the mouth is dry and sticky; the tongue is smooth, red, and shining; the saliva is acid, and sometimes contains sugar or lactic acid; the gums are softened and bleed easily; the teeth decay and fall from their sockets; the entire cavity of the mouth and pharynx exhibits evidences of subacute inflammation.

The exaggerated appetite that is experienced by many patients is due to the immense discharge of sugar, urea, and saline compounds. Sometimes intense gastralgia is experienced when the stomach is empty, and it can be relieved in no other way than by a hearty meal. Chronic catarrhal dyspepsia often exists, and the stomach becomes gradually dilated. In the later stages of diabetes, when the gastric glands have become atrophied, hydrochloric acid is often absent from the gastric juice; it is present in excessive amount when there is a ravenous appetite.

Constipation of the bowels is inevitable by reason of the excessive discharge of water with the urine and as a consequence of the reduction of biliary and intestinal secretion. Diarrhœa, however, sometimes occurs as a result of excessive eating or as a result of various nervous disturbances.

Perspiration is greatly diminished in the majority of cases: occasionally, however, it is profuse and highly charged with sugar and lactic acid. The skin is greatly irritated by such saccharine perspiration, and it consequently becomes the seat of various itching eruptions—*e. g.* pruritus, erythema, psoriasis, herpes, eczema, lichen, and xanthoma. The occurrence of peripheral neuritis, principally involving the vaso-motor nerves, sometimes determines the manifestation of symmetrical cutaneous œdema and erythema. Boils, carbuncles, and more or less extensive patches of gangrene are frequently witnessed during the course of diabetes.

The bodily temperature is generally subnormal, and if intercurrent febrile diseases occur they are accompanied by less elevation of the temperature than is usual in non-diabetic patients. This deficiency is due to the imperfect oxidation of the tissues and to the enfeebled state of the heart and other organs of circulation. The absorption of oxygen is reduced below the normal standard, and in the later stages of the disease the amount may fall to less than one-half the quantity that was absorbed during health. A corresponding reduction in the quantity of carbonic acid and other products of oxidation in the tissues follows as a necessary consequence.

The enfeebled condition of the nervous system is indicated by persistent muscular weakness, pain in the back, and cramps. Paroxysms of neuralgia are experienced in symmetrical parts of the body, especially in the sciatic nerves: they are often dependent upon peripheral neuritis that is excited by the excess of sugar in the blood. Various painful conditions are experienced in the joints, in the back of the neck, as well as in other parts of the body, and there is an inordinate sensibility to the influence of cold.

The patellar and other tendon-reflexes are often much diminished or totally abolished. Their complete absence is an unfavorable symptom. Occasionally there is an exaggerated condition of the patellar reflex; in such cases there is a lesion in the central nervous system to which the occurrence of diabetes must be referred, since glycosuria is almost always present whenever the fourth ventricle is invaded by diseases that involve the brain and spinal cord. In certain cases the symptoms of tabes dorsalis are developed during the course of diabetes, constituting what is termed pseudo-tabes. From genuine tabes this functional disorder may be distinguished by its rapid development and by its subsidence under the influence of remedies that favorably affect the course of diabetes. Trophic disturbances, such as perforating ulcer of the foot, shedding of the nails and of the hair, may be explained by the existence of peripheral neuritis. Brief apoplectiform attacks, followed by hemiplegia and attacks of syncope and vertigo, have been occasionally observed in connection with diabetic symptoms. Transient paralytic phenomena, usually restricted to single muscles or small groups of muscles like those which are connected with the eyeball, are sometimes witnessed. Transient ptosis and strabismus are thus sometimes produced. The phenomena of angina pectoris are sometimes counterfeited by paroxysms of asthma and of severe neuralgia in the præcordial region. Sometimes the occurrence of invincible somnolence marks a tendency to a condition of coma, while the failure of other bodily functions, intellectual debility, and mental disorder are sometimes observed.

In many cases the heart participates in the general disorder. In vigorous subjects the organ may become hypertrophied, but it is usually dilated when the health is deteriorated. In cases that are characterized by obesity fatty degeneration and arterio-sclerosis invade the heart and blood-vessels. Endocarditis is a frequent occurrence, and is always a dangerous symptom: it appears to be excited by a prolonged irritation of the endocardium with blood that is overcharged with sugar.

The breath frequently exhales the peculiar odor of rotten apples. Lobar pneumonia is often an element of great danger. Fibrinous pneumonia, with dilatation of the bronchi, induration of the pulmonary parenchyma, and copious purulent secretion, has been witnessed. Gangrene sometimes follows other inflammatory diseases of the respiratory organs. Pulmonary consumption is an exceedingly common complication of diabetes. Its commencement is very insidious; its progress is rapid; its symptoms are not conspicuous, but its destructive consequences are most remarkable. So insidious is the process that it is often unsuspected until unmasked by a physical examination of the thoracic viscera.

The function of vision is frequently impaired. Nearly all of the paralytic and inflammatory diseases of the eye may occur in connection with diabetes. Otalgia, otitis, and deafness sometimes result from diabetes, and a similar deterioration of the senses of smell and of taste is in like manner sometimes experienced.

The progress of diabetes is dependent upon the age of the patient and upon the type of the disease. Its course is very rapid among children, in whom it is usually manifested shortly before the age of puberty. In such cases the patient rapidly emaciates, and death generally occurs within two years from the commencement of the disease.

The conjunction of diabetes and pregnancy is a source of great danger to the patient: abortion occurs in one-third of the cases, and in one-half of them delivery is followed by death from pulmonary consumption or from coma. This malignant form of diabetes must not be confounded with the transient glycosuria that is sometimes witnessed during the period of lactation among young women.

Diabetes is liable to assume a malignant and rapid course as a consequence of excessive bodily or mental fatigue. When the disease occurs among elderly patients who manifest an arthritic predisposition and a tendency to corpulence, it may be protracted from fifteen to thirty years, but when it is accompanied by pancreatic disease and suppression of the pancreatic secretion, emaciation progresses rapidly, and the disease is terminated within eighteen months or two years.

Two strongly contrasted forms of diabetes have been noted in the preceding paragraph. The arthritic variety of the disease usually occurs in corpulent persons, who at first manifest transient glycosuria, and only after a considerable period of time does the disorder become permanently confirmed. The malignant form of diabetes commences suddenly in the midst of apparent health. It progresses rapidly, is characterized by emaciation and exhaustion; pulmonary consumption is a frequent complication, and death soon terminates its course. Unlike the arthritic variety, this form of diabetes is not traceable to heredity.

Injuries or concussion involving the head and the spinal column are sometimes followed by diabetes. When the symptoms are manifested immediately after the reception of an injury, recovery generally occurs in the course of three or four months; but if its development be delayed for some time after the accident, it pursues a tedious course that usually reaches a fatal termination.

Death from diabetes is frequently determined by the various suppurative or gangrenous processes that are excited by parasitic invasion of the body. In other cases it results from pulmonary consumption or from diseases of the liver or kidneys; but in many instances the conclusion is suddenly reached through the development of diabetic coma.

More than half the cases of diabetes terminate fatally in a condition of coma. Usually after some form of fatiguing exercise the patient becomes suddenly affected. Such an event is not uncommon after a journey or other extraordinary exertion. There is imminent danger of coma whenever the quantity of urine is diminished without any corresponding reduction in the amount of sugar that it contains. Intercurrent disorders that depress the nervous system are liable to induce a fatal termination in this way; and it has been thought that such a result may follow therapeutic measures which reduce the flow of saccharine urine.

Diabetic coma must not be confounded with that sudden cardiac collapse which sometimes prostrates the victims of diabetes. Such cardiac failure is a consequence of fatty degeneration of the heart, and it is generally followed by death in the course of twenty-four or forty-eight hours. It is a consequence of the general tendency to obesity and to fatty degeneration that is remarked in the arthritic variety of diabetes.

Diabetic coma usually occurs in connection with that type of diabetes which is characterized by rapid wasting and cachexia. The final prostration of the patient is preluded by a peculiar odor, like that of chloroform, exhaled with the breath and from the urine of the patient. The amount of urine is reduced, and the proportion of sugar that it contains is also diminished. In certain cases there is albuminuria, and the addition of chloride of iron causes the urine to exhibit a deep port-wine color. In many cases there is intense dyspnœa, even though the patient is able to maintain the recumbent position and though the respiratory movements are not accelerated. Auscultation and percussion indicate no morbid change within the thorax, and it is probable that the disturbance of respiration is due to a pathological state of the blood by which the respiratory centres in the medulla are thrown into disorder. The pulse remains nearly, if not quite, normal. The temperature of the body is gradually lowered, though it is sometimes elevated for a brief period at the commencement of the attack; there are nausea, vomiting, and diarrhœa; sometimes there is painful distension of the abdomen, but no symptoms of inflammation or of fever can be detected.

In certain cases the onset of the attack is attended with slight exhilaration and exaltation of the mental faculties, but this temporary excitement is soon merged in somnolence and coma. Convulsions are occasionally observed among young children.

The full development of coma is characterized by complete loss of consciousness: the patient lies motionless; the pupils are dilated, but react to changes in the intensity of light to which they may be exposed; the extremities grow cold; the temperature both of the surface and of the interior of the body is reduced; the muscles are completely relaxed; and death occurs in three or four days. Occasionally the attack is preceded by dizziness and sensations that recall the phenomena of alcoholic intoxication; in such cases traces of alcohol have been found in the urine.

Besides the acute form of diabetic coma a chronic variety of intoxication has been described. This is marked by a prolonged period of prostration, during which dyspnœa, abdominal distension, and the peculiar odor of acetone in the breath are the prominent symptoms. Sometimes these symptoms of intoxication appear and disappear at successive intervals for a considerable time before the incidence of the fatal attack.

Pathology.—It has long been known to physiologists that glycosuria can be artificially excited in the lower animals by puncture of the floor of the fourth ventricle just below the pneumogastric nucleus. A similar condition can be also produced by various injuries involving the cerebro-spinal axis between the optic thalami and its lower extremity. Lesions of the larger peripheral nerves and of the sympathetic ganglia may exercise a similar influence in certain cases, so that it is highly probable that diabetes is often thus originated by local injuries of the nervous system.

The reduction of Fehling's solution may be occasioned also by the action of various toxic substances, such as curare, carbon monoxide, nitrous oxide, chloroform, amyl nitrite, methyl-delphinine, hydrochloric and phosphoric acids, turpentine, corrosive sublimate, uranium nitrate, morphine, strychnine, chloral hydrate, phloridzin, and lactic acid.

It has been conclusively demonstrated that in a healthy condition of the digestive organs glycosuria is not produced by the excessive use of sugar and starchy food. It is therefore believed by many that the presence of sugar in the urine implies a chronic disorder of the alimentary canal and of the liver and pancreas. This hypothesis is supported by the observation that permanent diabetes can be produced in the lower animals by total extirpation of the pancreas. Such an observation, however, does not explain those cases of diabetes in which there is no disease of the pancreas and in which the nervous system exhibits disorder, nor does it explain those cases which appear to be connected with the arthritic diathesis. The hypothesis has been advanced that in a state of health the glucose that is discharged into the blood undergoes transformation and disappearance through the action of a ferment that is elaborated in the pancreas, and that when the function of that gland is arrested or abolished diabetes is developed by reason of the non-transformation of glucose in the blood.

According to another hypothesis, the liver is supposed to be the seat of disorder, rendering it impossible for the saccharine elements of the food to be stored up in that organ in the form of glycogen. Under such circumstances the blood must necessarily become overcharged with glucose, which is therefore eliminated through the kidneys.

Still another hypothesis represents the liver in a condition of over-excitement, producing an inordinate amount of glycogen and yielding to the blood a corresponding excess of glucose. Here, again, the presence of a special ferment has been invoked to account for these transformations.

According to a somewhat similar hypothesis it has been conjectured that the excessive transformation of glycogen into glucose takes place in the muscles rather than in the liver.

Another hypothesis is that the excess of glucose in the blood is due to retardation or failure of the process of oxidation by which it should be destroyed within the tissues. According to Pettenkofer and Voit, normal oxidation of the nitrogenous constituents of the body yields a certain amount of fat, but when the amount of oxygen is insufficient sugar makes its appearance instead of fat. It has been supposed that the retardation of oxidation takes place in the muscular substance of the body; but this hypothesis does not explain those cases of diabetes in which the urine contains no excess of nitrogenous waste.

Another theory is that the disorder at an earlier stage is due to changes in the passage of oxygen through the system. The accumulation of glucose in the blood has been ascribed to imperfect respiration and absorption of oxygen in the lungs; but considerable doubt is thrown upon this by the well-known fact that in pulmonary consumption the amount of oxygen that enters the blood through the lungs is greatly reduced, yet sugar does not appear in the urine; and in cases of diabetes that are complicated by similar pulmonary disorder the amount of sugar is not increased, but is diminished as the function of the lungs is impaired.

The excessive presence of sugar in the blood has by some been ascribed to insufficient alkalinity of the plasma. Recently it has been urged that various errors of nutrition are chargeable to a deficiency of selective energy on the part of the blood-plasma, and that when carbohydrates cannot be assimilated by the plasma sugar appears in the urine; while obesity results from the failure to assimilate fats, and an excess of nitrogenous excreta in the urine will in like manner follow a failure to dispose of ammoniacal nitrogenous compounds. Finally, it is affirmed that, if albuminous compounds that contain the elements of the three former alimentary substances cannot be disposed of by the plasma, albuminuria will be developed.

Out of these various fragmentary hypotheses certain authors have endeavored to construct a comprehensive theory that recognizes numerous types of diabetes. It has been maintained that minor forms of glycosuria are dependent upon a failure on the part of the hepatic cells to transform the hydrocarbonaceous constituents of the food into glycogen. In severe and malignant forms of the disease it is supposed that not only the hepatic cells, but all the constituent elements of the organism, have lost their power to assimilate the glucose that is conveyed to them by the blood. This, however, is merely a statement of a fact without any explanation of its cause.

No satisfactory explanation of diabetic coma has yet been advanced. It has been ascribed to the action of acetone, a substance that is undoubtedly present in the urine when the breath and the urine exhale an ethereal odor. This compound is, however, often present when no signs of diabetes or of coma are manifested, and it is frequently absent in cases of coma. When administered in large doses it produces no poisonous effects.

The failure in connecting the symptoms of diabetic coma with the action of acetone has led to the ascription of the symptom to the action of diacetic acid, but the same difficulties make their appearance in connection with this substance. Oxybutyric acid has been, therefore, by certain pathologists, designated as the active agent when the liquids of the body contain an insufficient amount of ammonia to effect its complete saturation.

Hypotheses that are so complicated and so numerous sufficiently prove our ignorance regarding the nature and causes of diabetic coma. The only thing that is clearly apparent is the fact that the condition is a result of intoxication with certain products of tissue-change within the body. If this fact be sufficiently recognized, the term "acetonæmia" may be employed to indicate the complex and unknown causes of diabetic coma, just as the analogous term "uræmia" is employed to express that unknown condition of retrograde metamorphosis that precedes and underlies the manifestation of renal coma.

Diagnosis and Prognosis.—The conspicuous symptoms of diabetes are polyuria, thirst, and a voracious appetite. The determination of the presence of sugar in the urine confirms the diagnosis. In many cases of diabetes, however, the urine is not increased and the appetite remains undisturbed; consequently the existence of glycosuria may remain long unsuspected unless chemical examination of the urine be made.

The presence of sugar in the urine may be determined by the use of the polariscope or saccharimeter. Such instruments, however, are not within the reach of the majority of practitioners. For these the well-known fermentation test affords an excellent means of detecting the existence of glycosuria. Numerous chemical tests have been described and employed, but from a clinical standpoint the most useful of them all is Fehling's solution. When properly prepared each cubic centimetre of the solution is reduced by 5 milligrammes of glucose. The proper application of the test consists in first boiling a drachm of the reagent in a test-tube: if its color and transparency remain unchanged, the reagent may be trusted for accurate results. Into the heated fluid the suspected urine should then be poured slowly down the side of the test-tube, so that it may flow upon the surface of the reagent. If any considerable quantity of sugar be present, the contiguous surfaces are clearly defined by a greenish film; but it rapidly changes to yellow, orange, and finally to a red color as the process of reduction extends itself. When the quantity of sugar is insignificant it will be found necessary to boil the contents of the test-tube for a few seconds. Dilution of the solution with about four times its bulk of water adds greater delicacy to the test.

The presence of albumin in the urine will prevent the reduction of the reagent, even though sugar be present. Under such circumstances it is necessary to effect its removal: this may be accomplished either by coagulation under the influence of heat or by precipitation with a solution of the subacetate of lead. The salts of ammonium should be removed by heating the urine with a little caustic soda, after which the test may be employed as usual. Uric acid and the urates, if precipitated, must be removed by filtration or by the addition of subacetate of lead, which also at the same time effects the elimination of albumin.

The inhalation of chloroform or nitrous oxide and the administration of chloral hydrate are frequently followed by apparent glycosuria, but this is transient, and does not persist after the elimination of the drugs.

The mere presence of sugar in the urine must not be taken as evidence of diabetes. Having determined the existence of glycosuria, its permanence must be ascertained by repeated examination. If dependent upon faulty digestion or upon the excessive use of saccharine and starchy food, sugar will be found in the urine only during the period of digestion, and its quantity will be commensurate with the amount of carbohydrates in the food. The urine should be examined twice during a period of twenty-four hours, the first specimen being taken immediately after arising in the morning, while the second should be taken three or four hours after a meal. In connection with these tests the condition of the digestive organs should be carefully observed, since the existence of intestinal indigestion adds to the dangers of the disease, being usually dependent upon serious disturbance of the pancreatic function. Albuminuria is also a dangerous complication. The amount of urea and other excrementitious substances in the urine should be ascertained, since they afford an accurate measure of the amount of retrograde metamorphosis in the tissues. The hereditary tendencies and diathetic characteristics of the patient should also be ascertained, since diabetes occurring in arthritic subjects is less formidable than when occurring in scrofulous, tubercular, and cachectic patients. The concurrence of diabetes with obesity, gout, lithiasis, and other arthritic disorders is highly significant, and necessitates the adoption of a special diet and habits of life. When diabetes is dependent upon sedentary habits and excessive eating it is less rebellious to treatment than when it is associated with hereditary arthritic tendencies.

When diabetes can be traced to causes of a psychical or neurotic character or to shocks and injuries that affect the nervous system, the prognosis will be largely influenced by the prospect of recovery from such injuries or perversions of nervous function. The early appearance of glycosuria under such circumstances is more favorable than its later development. The disappearance of the tendon-reflexes is an unfavorable symptom, and so also are insomnia, loss of sexual appetite, failure of memory, and deterioration of the special senses. Diabetic patients should be frequently weighed, in order to ascertain whether they are losing flesh. The lungs should also be examined at stated intervals, since latent tuberculosis is not uncommon. The condition of the skin should be noted, since its disorders are indicative of defective nutrition.

Treatment.—The treatment of diabetes is comprised chiefly in the regulation of the diet. The carbohydrates, sugar and starch, should as far as possible be excluded from the food, since the greater portion of the glucose that enters the blood is derived from those substances. As a substitute for sugar, saccharin is to be recommended, but its taste is to many patients so disagreeable that its utility is considerably restricted. Glycerin may be sometimes used in its place. All fruits and vegetables that abound in starch and sugar must be forbidden —viz. potatoes, rice, all compounds of flour or starch, beans, peas, lentils, chestnuts, turnips, radishes, grapes, plums, apricots, pears, apples, melons, figs, strawberries, cherries, gooseberries, raspberries, carrots, beets, onions, tomatoes, and asparagus. The following articles may be allowed: All kinds of butcher's meat, game, poultry, fish, oysters, clams, eggs, bacon, butter, fats, glycerin, spinach, chiccory, cabbage, cauliflower, water-cress, lettuce, and other salad plants. The different kinds of cheese, olives, nuts, unsweetened chocolate, and table salt (for which may be substituted tartrate of sodium, citrate of sodium, and phosphate of calcium) are permitted. All saccharine beverages must be forbidden—viz. lemonade, champagne, beer, cider, ginger ale, and other aërated drinks, and milk; skimmed milk has been recommended by many physicians, but its use is not often attended with satisfactory results.

Water, and coffee or tea without sugar, may be allowed. Alcohol should be avoided on account of its injurious influence upon the liver. The patient should be permitted to drink water freely, since it is needed for the elimination of sugar, and, if withheld, will be absorbed from the tissues themselves. Alkaline waters like Vichy water and Londonderry lithia water, and alkaline calcic waters like Waukesha and Apollinaris water, are especially useful.

Bread made with gluten flour has been extensively employed in the treatment of diabetes, but it is exceedingly unpalatable, and is liable to interfere with the digestion. It also contains so much starch that it is inferior to potatoes as an article of diet. Potatoes should be allowed in moderate quantity as a substitute for bread, since they contain carbonate of potassium, a salt that greatly aids the assimilation of sugar. The use of almond flour in the form of cakes made with eggs and cream has been recommended, but the article is too expensive for general use.

Simple cases of glycosuria can be cured, according to the method of Cantani, by restricting the patient to a diet of fat meat; but in genuine diabetes a more liberal dietary must be permitted, for fear of other complications, among which many observers enumerate the predisposition to diabetic coma. According to Naunyn, there are three forms of diabetes: the severe, the moderate, and the mild form. In severe cases of the disease, so long as the urine is not reddened by the addition of perchloride of iron—a symptom that indicates a tendency to diabetic coma—he recommends a restricted diet of fat meat. In moderate cases, under a flesh diet, after the disappearance of sugar from the urine, he permits the use of eggs, milk, and a small quantity of bread. In the mild forms of the disease a more liberal diet is allowable, but the reappearance of sugar in the urine should be followed by greater caution with regard to the dietary. Salads, string-beans, mushrooms, pears, and apples may be allowed. Beer is prohibited, and milk may be used only when it does not increase the quantity of sugar in the urine. In mild forms of diabetes the daily ration consists of sixteen to eighteen ounces of meat; one to three ounces of bread; three to four ounces of vegetables twice a day, or an equal quantity of salad or apples. Active exercise, gymnastics, and massage are to be encouraged, since they aid the processes of oxidation in the tissues. For the same reason warm baths should be taken three times a week. The clothing must be warm and the patient must be protected from cold, since a chill is always injurious in cases of diabetes. All violent emotions and excessive fatigue must be avoided. During the winter season a residence in a warm climate should be secured if possible.

The pharmaceutical treatment of diabetes consists chiefly in the employment of remedies that favor the process of oxidation and restrict the liberation of glucose from the liver. The alkalies are especially useful in recent cases of the disease when the patient is not too far advanced in years and is vigorous, fleshy, and plethoric, with a predisposition to obesity, gout, and rheumatism. They are also useful when the urine contains an inordinate quantity of uric acid: such patients may be encouraged to drink freely of lime-water. Magnesia; liquor ammoniæ, of which six drops may be taken in water three times a day; carbonate of ammonium, from fifteen to eighty grains per diem; thirty to sixty grains of bicarbonate of sodium three times a day; the citrate, tartrate, malate, and bicarbonate of potassium, sodium, or lithium,—are all valuable alkaline salts. The utility of Vichy water and of similar mineral waters is due to the alkaline salts which they contain. Bicarbonate of sodium is prescribed in cases of diabetic coma by those who adopt the hypothesis of its dependence upon an excess of acids in the tissues. The use of alkalies and of alkaline mineral waters also serves to diminish the amount of uric acid and sugar when there is an inordinate production of urea and of other nitrogenous excreta. Under the influence of such alkaline remedies digestion improves, emaciation is arrested, and obesity is reduced. When, however, there is conspicuous evidence of retardation in the process of oxidation, alkaline remedies should no longer be employed.

Excessive hunger and thirst are most efficiently relieved by the exhibition of opium and its derivatives. Under their influence the amount of sugar in the urine is largely decreased. Codeine is particularly useful when the amount of nitrogenous excreta is excessive; the drug may be given in large doses if combined with strychnine for the purpose of sustaining the processes of respiration. Belladonna is sometimes of service in recent cases, but it is useless and often injurious in advanced cases, especially when the kidneys have undergone degeneration. Nux vomica and strychnine are useful in cases that are characterized by exhaustion and by enfeeblement of the visual, digestive, and generative organs. Laxatives and purgatives are liable to increase the amount of glycosuria; hence they must be administered with great moderation.

Thirst and polyuria are diminished by the administration of valerian, a remedy that is exceedingly useful when the amount of urea is excessive. Arsenic and its compounds have been often prescribed, but their effects are seldom satisfactory. The compound tincture of iodine, in doses of five to twenty drops, largely diluted with water, may be given before each meal. Mercury is useful in cases that are complicated with syphilis. Many other alterative remedies have been employed, such as mineral acids, salicylate of sodium, carbolic acid, cocaine, astringent substances, ferments like yeast, diastase, and pepsin, jambul, permanganate of potassium, peroxide of hydrogen, ozonized ether, inhalations of oxygen, etc.; but none of them can be relied upon for permanent and satisfactory results.

Remedies that are addressed to the nervous system often produce considerable temporary effects. Forty-five grains *per diem* of antipyrine cause a rapid reduction in the amount of sugar that is discharged with the urine; but the drug is useless in cases that are complicated with tuberculosis or characterized by progressive emaciation. The valerianate and other salts of quinine are often administered in doses of three to ten grains a day. Bromide of potassium is often useful when diabetes is associated with diseases of the medulla oblongata. The hypodermic injection of ergotin and other liquid preparations of ergot has been practised with indifferent success. Electricity has been employed in every possible way, and under its influence glycosuria is sometimes temporarily diminished. Blisters and other counter-irritants should be avoided for fear of gangrene at the point of vesication. Indigestion and anæmia require the ordinary treatment for such complications. Carbolic acid and lactic acid have been administered for the purpose of favorably influencing the processes of digestion and assimilation, but their ability is decidedly questionable. About half the cases of diabetes may be temporarily cured, but the great majority are liable to relapse and to fatal termination at last.

POLYURIA.

BY HENRY M. LYMAN.

Definition.—Polyuria is a constitutional disease characterized by an increased quantity of urine, thirst, excessive craving for liquids, and considerable variation in the proportion of the soluble constituents of the urinary secretion. A small quantity of inosite or muscle-sugar sometimes appears in the urine. In certain cases the nitrogenous excreta are discharged to an inordinate degree, and in others the saline constituents are abnormally increased. When albumin appears in the urine of polyuria, it is owing either to the existence of interstitial nephritis or to the occurrence of general neurotic or arthritic disorders.

SYNONYMS.—Diabetes insipidus; Diuresis; Hyperuresis; Hydruria; Polydipsia; Urinæ profluxio; Einfache zuckerlose Harnruhr.

Etiology.—Polyuria is usually a disease of middle life, though it is sometimes encountered in early childhood. It is more frequent among men than among women, in the ratio of two or three to one. Hereditary conditions exercise an important influence over its development, and in certain families polyuria and saccharine diabetes alternate with each other. Like glycosuria, polyuria may follow every kind of injury or disease of the nervous system. It often occurs as a sequel of the infective diseases and of chronic intoxication with lead or alcohol. It is not uncommon as a consequence of exposure to cold or to excessive heat, or of a sudden refrigeration of the body, or any other form of shock or violent disturbance of the nervous system.

Symptoms.—Polyuria is characterized by an excessive discharge of urine. The quantity of liquid that is voided may be increased two- to tenfold, and the patient is annoyed by a frequent desire to urinate. The urine is light yellow or nearly colorless; its reaction is acid, though it rapidly becomes neutral or alkaline after standing in the open air; and the specific gravity seldom reaches 1010. Though relatively diminished, the solid constituents of the urine often exhibit an absolute increase, in consequence of the excessive amount of fluid that is discharged. Sugar is occasionally present for a short time. If albumin be detected, its presence must be referred to an inflammatory condition of the kidney when it is not dependent upon hæmorrhage or inflammation in the urinary ducts below the kidney.

Inordinate thirst is commonly experienced during the course of polyuria. The mouth and throat become dry and sticky; the tongue is red and glazed; perspiration diminishes; the bodily temperature frequently falls below the normal standard. If the disease occur during childhood, it interferes with the growth of the body; the appetite is irregular and subject to various perversions. The process of gastric and intestinal digestion is usually disordered. In certain cases there are headache, dizziness, and diminished power of intellectual action. In severe cases mild delirium may be observed. The sixth nerve is frequently paralyzed, and sometimes other cranial nerves are similarly affected. The reflexes sometimes disappear, hæmorrhage is occasionally observed in the retina, and neuro-retinitis, together with atrophy of the optic nerve, may occur: hemianopsia and amblyopia are frequent incidents, but the development of cataract is a rare event.

The nitrogenous constituents of the urine are greatly increased in quantity. In many cases the symptoms closely resemble the characteristic features of saccharine diabetes. The amount of urine is greatly increased; there are intense thirst and voracious appetite, progressive emaciation, dryness of the skin, intolerable itching, disturbance of the nervous system, and in many instances a rapid development of phthisis. Though heavily charged with urea, the urine does not contain sugar.

The onset of the disease is sometimes very abrupt, though it usually commences in a gradual and insidious manner. The total amount of urine is always less than the aggregate of the liquids that are drunk; its quality varies according to the amount of urea that is discharged. Its color is light yellow, except when temporarily concentrated by remedial measures. The liquid is acid when first voided, but it soon becomes turbid and ammoniacal, in consequence of the presence of mucus and epithelial cells that are discharged from the urinary passages as a consequence of their irritation by the excreta that are held in solution. The specific gravity is usually but little above that of distilled water, yet it may vary from 1002 to 1050. In the sediment may be found urates, uric acid, and occasionally oxalate of calcium. The daily amount of urea varies from an ounce and a half to three ounces, but it often measures much above or below these figures. The amount of uric acid seldom exhibits any considerable increase, but there is an excessive discharge of other nitrogenous excreta and of the chlorides and phosphates. A considerable portion of this excessive waste must be referred to the enormous quantity of food that is consumed by many of these patients.

The course of the disease is marked by great exhaustion and progressive debility. There is rapid emaciation; the extremities are frequently cold and livid, in consequence of feeble action of the heart; the temperature often falls below the normal figure; active exercise is rendered impossible by reason of muscular weakness; headache is frequent, and may become a constant experience; hæmorrhage, itching of the skin, and other perversions of sensation involve the external surface of the body and the mucous membranes; degradation of the special senses and amblyopia, with or without visible lesions, are common incidents; impotence is an early and almost universal symptom; insomnia and vertigo frequently occur; the intellectual faculties are gradually extinguished; tremors, spasms, or general convulsions are sometimes witnessed, and death may be preceded by coma.

The duration of the disease is usually quite protracted. Emaciation is frequently succeeded by dropsy. As the appetite and power of digestion fail, the amount of urea diminishes. Death is caused either by exhaustion, hæmorrhage, gangrene, convulsions, coma, or pulmonary consumption. Only in rare instances has recovery resulted from the intercurrence of an acute infective disease or through the action of valerian or opium.

Diabetes insipidus, with phosphaturia as a prominent symptom, generally accompanies tuberculosis or some disorder of the nervous system. The urine contains ammonio-magnesian phosphates: if urates and oxalates are present in excessive quantity, an iridescent film appears upon the surface of the liquid after it has been exposed to the open air for a considerable time. The disease is sometimes connected with glycosuria. Many of the symptoms of saccharine diabetes are experienced, and sugar appears at intervals in the urine. The excessive elimination of phosphates is supposed to be dependent upon the splitting up of glucose within the organism into lactic acid, which renders the phosphates more soluble and diffusible, so that they quit their normal position in the tissues and are discharged with the urine. When the disease is manifested in children and young people, it is frequently associated with the presence of oxalates and an excessive quantity of uric acid in the urine. Traces of albumin are also sometimes apparent. All these symptoms indicate the existence of the arthritic diathesis, and serve to illustrate the near relationship of the disease to gout and other arthritic disorders.

Pathological Anatomy.—Polyuria is unaccompanied by any characteristic pathological changes. In certain cases a proliferation of the connective tissue and consequent degeneration of the nervous elements in the solar plexus has been described. The kidneys are frequently enlarged and distended with blood. Sometimes the uriniferous tubules are dilated, and their epithelium may exhibit fatty degeneration; but the symptoms are not explained by these variable and almost accidental changes, nor do they throw any light upon the nature and cause of the disease.

Diagnosis.—Polyuria can be easily recognized if a chemical examination of the urine be added to a review of the symptoms. From saccharine diabetes and from chronic nephritis the disease may be distinguished by the results of urinalysis. Transient polyuria and primary polydipsia may be distinguished by the history of the patient, and by the fact that in such disorders the excessive flow of urine is not constant, but is immediately connected with the occurrence of thirst and the consequent gratification of the appetite for food and drink. Oxaluria, phosphaturia, and other urinary disorders must be differentiated from polyuria by the results of urinalysis taken in connection with the characteristic symptoms of disordered nutrition.

Prognosis.—Recovery is rarely witnessed, though the condition of the patient may be greatly ameliorated, if not permanently restored to health, by treatment. Rapid emaciation and progressive exhaustion are unfavorable symptoms, though in many cases life is prolonged for a number of years.

Treatment.—Uncomplicated polyuria is benefited by the administration of antipyrine in large doses; fifteen grains should be given every four hours. Lead and opium have been employed, but they are less efficacious, and whatever benefit is derived from their use is principally due to the opiate. The diet must be restricted, and it should be of the most nutritious character; thirst is greatly relieved by acidulated drinks. When the urine contains nitrogenous excreta in excess the patient should make free use of meats and starchy food. Quinine, arsenic, and codeine, especially when administered in combination with strychnine, exert a favorable influence upon the processes of nutrition. Valerian is one of the most useful remedies, and it should be given in large doses: an ounce of the extract may be given in divided doses during each successive period of twenty-four hours if tolerated by the stomach. Corresponding doses of the powdered root are recommended by many physicians, but these large doses are not always endurable. Insomnia, delirium, and irritable weakness of the nervous system are most effectually relieved by opiates: codeine is one of the most efficient; it may be given in doses of one grain every four hours. Mercurial inunctions and iodide of potassium are only to be employed in syphilitic subjects.

Phosphaturia, when dependent upon tuberculosis, requires particular attention to the pulmonary complication. Bromide of potassium and other neurotic remedies are useful in cases that are characterized by excitement of the nervous system. Alkalies and alkaline salts must be administered whenever there is an excess of acid in the alimentary canal. Compensation for the excessive waste of phosphates must be secured through the use of food that is rich in phosphates: for this reason whole-meal flour should be preferred to the ordinary bolted flour, and eggs and fish may be given with great advantage.

RHEUMATOID ARTHRITIS.

By HENRY M. LYMAN.

Definition.—Rheumatoid arthritis is a chronic disease involving the articulations of the body, especially those of the extremities: it is characterized by fibrillation and erosion of the cartilaginous surfaces and by the formation of osteophytes around the ends of the bones. The disease progresses without fever, is characterized by painful paroxysms at irregular intervals of time, and results in permanent deformity of the affected joints, especially involving those of the fingers and toes. The disease is usually primitive in its origin and course, but it sometimes occurs as a secondary consequence of gout, acute articular rheumatism, or gonorrhœal arthritis. Occasionally it invades the larger joints, preferably the hip-joint, at a late period of life, often as a consequence of some trifling injury that has been sustained by the structures that constitute the affected articulation. Of this variety of the disease the morbus coxæ senilis furnishes the best example.

Synonyms.—Nodosity of the joints (Haygarth); Chronic rheumatic arthritis or rheumatic gout (Adams); Arthritis rheumatismo superveniens (Musgrove); Goutte asthénique primitive; Arthritis pauperum; Arthritis sicca; Usure des cartilages articulaires (Cruveilhier); Arthrite chronique (Lute); Progressive chronic articular rheumatism; General and partial chronic osteo-arthritis; Arthritis deformans; Polyarthrite déformante; Pseudo-rheumatisme noueux.

Etiology.—Hereditary influences are not unimportant in the determination of rheumatoid arthritis, yet they seem to be less potent than in rheumatism and gout. Among the cases collected by Archibald Garrod, 43 per cent. of the patients could trace articular disease, chiefly of a gouty character, among their ancestors, while only 12 or 13 per cent. could refer to rheumatism among the antecedents in their family history.

Rheumatoid arthritis is far more commonly experienced by women than by men. Among 500 cases collected by Garrod, only 89 occurred among males. Uterine disorders and the suppression of menstruation appear to exert a great influence upon the development of the disease.

Rheumatoid arthritis is most frequently experienced during the fifth decade of life, though it is sometimes observed in childhood and youth. Among females the number of cases increases with each period of five years until the age of forty-five. After fifty years of age females are less frequently attacked, while among males the corresponding degree of liability is not reached until the seventieth year is passed.

In a large proportion of the female patients who are afflicted with rheumatoid arthritis the commencement of the disease dates near the menopause. It is also frequently associated with the occurrence of uterine diseases, and it is probable that in many instances these are dependent upon the same causes that produce arthritic disease.

The dependence of rheumatoid arthritis upon a condition of lowered vitality is also illustrated by the effects of mental disturbance and depressing emotions. Anxiety, worry, and fatigue are frequent antecedents of the first symptom of arthritis, and during its course any unfavorable incidents of this kind are liable to be followed by an exacerbation of the articular disease. It is through the depressing influence of fright and other violent nervous shocks that the connection between nervous depression and rheumatoid arthritis is frequently exhibited.

Long-continued exposure to wet and cold forms an important predisposing cause of rheumatoid arthritis, especially when associated with want and privation. It is for this, among many other reasons, that the disease is so frequently observed among the poor. When the disease has been fully established fresh exposure to cold and damp is always attended by an increase of pain in the affected joints.

The effect of local injury as an exciting cause of rheumatoid arthritis is most conspicuous in the production of morbus coxæ senilis rather than the ordinary forms of the disease, but it is undoubtedly true that injuries of the smaller articulations are sometimes followed by the characteristic deviations, deformities, and pain of rheumatoid arthritis. I have this very day seen an old man of seventy-three years presenting in his right hand all the characteristic symptoms of rheumatoid arthritis, which he attributes to an injury of one of the knuckles twenty years ago. The disease is sometimes experienced as a sequel of acute articular rheumatism, gout, or gonorrhœal arthritis.

A liberal dietary and the use of alcoholic beverages seem to exercise no influence in the production of rheumatoid arthritis: it is a disease that is benefited rather than caused by a generous supply of food.

The antecedents of rheumatoid arthritis are often closely related with scrofula, which is a diathetic representative of the consequences of physiological privation and social misery. The disease is clinically related with those arthropathies which are manifested in the course of tabes dorsalis, hemiplegia, and paralysis agitans.

Pathological Anatomy.—The changes that are encountered in the articular structures as the result of rheumatoid arthritis consist largely in an exaggeration of the ordinary senile changes that are observed as a result of impaired nutrition in old age. It is probable that the inflammatory changes that are witnessed are the secondary consequences of the deteriorated nutrition which constitutes the essential cause of arthritic change.

At an early stage in the course of the disease the articular cartilage assumes a velvety appearance as a consequence of fibrillation of the intercellular substance, associated with disappearance of the cartilage-cells. The disappearance of the cellular elements of the cartilage is preceded by their multiplication and aggregation in compound capsular masses, which finally become ruptured and discharge their contents into the articular cavity. The fibrillated interstitial substance undergoes a mucous degeneration which facilitates the process of erosion and removal of the remains of the cartilage upon the articular extremities of the bones. At the exterior margin of the articular cartilage the compound capsules cannot discharge their contents into the cavity of the joint, since the synovial membrane prevents access to its interior. The thickened margin therefore protrudes, and becomes transformed and deformed by the production of ecchondroses around the periphery of the articulation. These ecchondrosal enlargements are finally ossified, and constitute the osteophytes which enlarge and deform the extremities of the bones that enter into the formation of the affected joint.

The destruction of the articular cartilage leads to a peculiar infiltration of the denuded extremity of the bone, giving to its substance an ivory-like density and hardness. This process of eburnation is intensified by the effects of mechanical pressure and by the modifications of nutrition that are manifested in the osseous tissue itself. By these processes, combined with the formation of osteophytic growths around the head of the bone, great destruction and deformity of the normal articular structures are effected. Great impairment of the mobility of the affected joint is thus produced, but true osseous ankylosis never occurs except in the articulations of the spine. When the disease is assimilated to the senile changes of old age, the affected bones usually become spongy instead of undergoing condensation, and the spaces that are thus formed in the osseous tissue are filled with a marrow-like substance.

The synovial fringes present the appearance of tufts in which are developed masses of fat or of cartilage. These little cartilaginous projections are sometimes separated from the membrane in which they were formed, and lie disconnected in the articular cavity. The ligamentum teres of the hip-joint and the long tendon of the biceps muscle in the shoulder-joint undergo absorption as the disease progresses. Sometimes the synovial capsule is greatly distended with fluid, though in many cases the amount of liquid is greatly reduced, if not entirely removed, so that the articular surfaces creak and grate when moved upon one another.

The muscles in the neighborhood of the affected joint when they undergo atrophy exhibit the partial and progressive atrophic changes which are characteristic of progressive muscular atrophy that is dependent upon central nervous disease. In certain cases the peripheral nerves exhibit unmistakable evidences of inflammation, but nothing has yet been discovered in the spinal cord that can explain the occurrence of articular disease.

The majority of the visceral lesions that have been discovered after death may be ascribed to intercurrent diseases rather than to rheumatoid arthritis. Pulmonary tuberculosis and chronic interstitial nephritis are among the most common of the changes that are thus brought to view. Cardiac lesions are rare, and are probably due to atheroma or to renal disease or to previous attacks of articular rheumatism.

Symptoms.—Painful enlargement of the phalangeal joints of the extremities is usually observed as the earliest characteristic symptom of rheumatoid arthritis. But in many cases this phenomenon is preceded by premonitory symptoms which take the form of numbness or tickling sensations in the fingers and toes. Palpitation of the heart may be sometimes observed; the rate of the pulse is often accelerated and its tension is increased. An irregularly freckled appearance of the skin upon the hands, neck, and other parts of the body is sometimes observed as one of the earliest symptoms of the impending disease. Local perspiration upon the palms of the hands or upon the forehead is sometimes noticed. Neuralgic pains, muscular cramps, and an increase of irritability in muscles that have already become partially atrophied are frequently experienced.

Rheumatoid arthritis sometimes commences abruptly, with acutely painful swelling of the small joints. The articular swelling is restricted to the synovial capsule, and, though extremely obstinate, the disease is unaccompanied by the profuse sweat and liability to endocardial or pericardial inflammation that are witnessed in acute rheumatism. If there be any fever, it is very moderate and not of long duration: the articular swelling results in the formation of osteophytes around the ends of the bones, producing characteristic deformity of the joints. Among young children the disease sometimes invades many joints in rapid succession, causing their enlargement and atrophy of the muscles by which they are moved. The development of the disease when it occurs among children is much more rapid than when it attacks adults and old people. It is also sometimes characterized by great swelling without corresponding deformity, whereas among elderly people it progresses slowly, with but little pain, and results in well-marked deformities.

Rheumatoid arthritis invades the small joints of the fingers more frequently than any of the other articulations. Sometimes the disease is limited to the distal articulations of the fingers, producing those enlargements that were first described by Heberden, and are called, after him, "Heberden's nodes." In many instances a regular progression of the disease from the distal articulations may be witnessed as the disease successively invades the joints of the knuckles, the wrists, the elbow, and the shoulder. A similar tendency is frequently observed in the lower extremities, but sometimes no such regular progression can be discovered.

The invasion and progress of the disease are characterized by remarkable symmetry in development. Not only are the corresponding joints upon the two sides simultaneously invaded, but the corresponding portions of the articular structures are similarly affected. Occasionally, however, the disease is more severely experienced upon one side than upon the other, and its unilateral manifestation is sometimes witnessed. The articulations of the cervical spine are sometimes stiffened by the invasion of rheumatoid arthritis, and in 25 per cent. of the cases the temporo-maxillary articulation, which usually escapes other forms of arthritis, is invaded, together with other joints, in the course of rheumatoid arthritis. A complete ankylosis of this joint sometimes occurs, but more frequently it is only stiffened, so as to render movements of the jaw difficult and somewhat painful.

The earliest evidence of deformity consists in an enlargement of the ends of the bones that form the affected joint. The synovial capsule and the bursæ in the neighborhood of the joint are also distended with fluid. When the distal phalangeal articulations are involved, constituting what is known as Heberden's nodes, the terminal phalanx is sometimes pushed toward the radial side of the hand by reason of a bony growth upon the ulnar side of the articulation. The other joints of the fingers do not exhibit such radial deflection, but the fingers themselves are usually drawn toward the ulnar side of the hand, producing lateral distortion of the knuckles, which also become enlarged and knobbed by the changes that take place in the extremities of the metacarpal bones. This ulnar deflection is somewhat aided by the action of the extensor tendons, but it is chiefly due to a relaxation of the articular ligaments. When the wrist is invaded the enlargement of the articular extremities and the thickening of the sheaths of the tendons cause the forearm to present an almost uniform size from the elbow to the wrist. The cutaneous investment of the affected joints usually retains its natural color, though in early stages of the disease a moderate degree of redness may be sometimes observed. Flexion of the joints causes a peculiar cracking sound, and in old cases of the disease a grating sound is audible when the eroded ends of the bones are caused to move upon one another.

As the disease progresses the muscles by which the affected joint are moved undergo atrophy, so that an additional cause of deformity is thus established. The extensor muscles are most conspicuously affected, the tendon reflexes are increased, and the condition of the muscles and nerves points to a nervous origin of atrophy rather than to simple disuse of the affected muscles as its cause. In the majority of cases the reaction of degeneration is not manifested unless an extreme degree of atrophy has been reached. In some cases the tendon-reflexes are so considerably exaggerated that a tap upon a single tendon will produce reflex contractions throughout the entire limb or even involving the whole body. Ankle-clonus can be excited in many cases. These facts seem to indicate a central cause in the lateral columns of the spinal cord similar to what may be observed in certain spinal diseases.

The increase of nervous excitability is further indicated by a tendency to tonic and clonic spasms in different muscles of the body and extremities. These contractions are often unattended by pain, but when they invade the muscles of the affected extremities they serve to increase the deformity and displacement of the diseased articulations. In advanced cases of the disease the atrophied muscles become permanently contracted, and thus maintain the deformities that have been gradually produced by the various causes previously enumerated. Two principal types of deformity may be observed in the fingers and toes. The first has been characterized as the type of extension. In this the distal phalanges are more or less flexed upon the middle phalanges, which in their turn are in a state of hyperextension, while the proximal phalanges, again, are in a position of partial flexion upon the metacarpal bones, and the bones of the hand and wrist are slightly flexed upon the forearm. In a second class of cases, characterized by flexion, the distal phalanges are in a state of hyperextension; the middle phalanges are partially flexed, while the proximal phalanges are hyperextended, and the bones of the hand and wrist are considerably flexed upon the forearm.

In the production of these types of deformity the condition of the interosseous muscles plays an important part. By their spasmodic contraction the deformities of the type of extension are produced, while the type of flexion exists when they are weakened.

The skin that covers the affected joints, especially when the fingers are involved, frequently exhibits dystrophic changes, of which the phenomenon of "glossy skin" is one of the most common. The nails frequently become brittle and longitudinally curved. Ulceration of the fingers and atrophy of their pulp are not uncommon features. In the lower extremities œdema sometimes exists. Occasionally fibrinous nodules may be discovered among the muscles of the upper extremities, but these must not be mistaken for the nodules of articular rheumatism.

Pain in the joints is increased by movement and by the warmth of the bed at night; neuralgic pains and painful cramps are not uncommon. Besides these, the occurrence of inflammation in the spinal articulations causes painful compression of the spinal nerves. Such attacks are sometimes followed by temporary loss of sensation and power of motion.

Opinions have been considerably divided regarding the occurrence of visceral lesions involving the heart, the pericardium, and other internal organs, in connection with rheumatoid arthritis. The majority of English physicians incline to the belief that such disorders are referable to rheumatism as a complication, and that the lesions of rheumatoid arthritis are nearly always, if not constantly, restricted to the articular structures of the body. Among the French physicians the belief prevails that endocarditis and pericarditis may occur during the course of rheumatoid arthritis, just as they occur in connection with genuine rheumatism, but this connection is much less commonly witnessed in the arthritic disease than in rheumatic cases.

Inflammation of the conjunctiva, iris, and sclerotic capsule of the eye is occasionally observed, but in a certain proportion of such cases the cause may be referred to gout or to gonorrhœal rheumatism. In a considerable number of cases deafness results from rheumatoid arthritis involving the articulations of the small bones within the ear.

The general health is not always greatly deteriorated, though in chronic cases many patients suffer from confinement to the house and from the want of exercise that follows extensive injury of many joints. Some, however, remain crippled and incapable of any considerable locomotion without complaint of serious ill-health, unless from some accidental intercurrent disease. The chronic course of the disease is usually unattended by fever, though a slight elevation of temperature is sometimes observed in acute cases. The urine exhibits a diminution in the amount of phosphates, and there is great variability in the quantity of urea and uric acid that are contained in the urinary excretion.

Rheumatoid arthritis is not in itself a fatal disease, since patients frequently reach great age, though sufferers with articular deformity and disability for many years. Tuberculosis of the lungs and chronic disease of the kidneys are among the most frequent causes of death, but the fatal termination is often due to other ordinary intercurrent diseases.

When rheumatoid arthritis follows an attack of acute articular rheumatism, it pursues the same course that is exhibited when it occurs as a primary disease. Cardiac lesions are rather more frequently experienced in such cases than in those forms of the disease which commence independently of rheumatism. It appears probable that the pre-existence of a rheumatic affection of the joints and other tissues of the body predisposes them to a greater degree of nutritional disturbance when they are subsequently exposed to the causes of rheumatoid arthritis. That there is no necessary connection, however, between rheumatism and rheumatoid arthritis seems to be indicated by the fact that the rheumatoid disease may follow gonorrhœal rheumatism and gout as well as acute articular rheumatism. It is probable that many morbid causes which operate upon the articular structures may thus become predisposing causes of rheumatoid arthritis.

The connection between previous disturbance of the nutrition of a joint and subsequent rheumatoid inflammation receives additional illustration from the fact that the form of rheumatoid arthritis which involves a single joint or a few of the joints may be often traced to a slight injury of the subsequently affected articulation. These monarticular forms of rheumatoid arthritis produce alterations and deformities that are in their character identical with the changes that are developed in the polyarticular forms of the disease. The only conspicuous difference lies in the fact that the monarticular variety is manifested in large joints, and does not tend to invade all the articulations of the extremities and body. It is a single hip-joint or shoulder-joint that is usually affected, and the disease is experienced more frequently by men than by women.

When the hip-joint is invaded the commencement of the disease is indicated by a dull gnawing pain in the neighborhood of the joint and in the knee. Rotation of the head of the femur in the acetabulum becomes more and more difficult, and the affected thigh can only with difficulty be raised and crossed over the other. Stiffness of the tissues around the hip-joint increases, though pressure upon the trochanter is not particularly painful and the patient continues to move about with some degree of freedom. As the disease advances, however, the muscles of the hip undergo atrophy, and in many instances there is similar wasting of the muscles of the thigh. The knee-jerk is somewhat increased; the head of the femur is gradually eroded and absorbed, so that the limb becomes considerably shortened; sometimes large cysts are developed outside of the joint, often without any apparent connection with the articular cavity. In advanced stages of the disease, after destruction of the articular surfaces, a grating sound is audible when the thigh is moved upon the pelvis. Sometimes the monarticular form of the disease is merged in a subsequent development of polyarticular deformities of the same nature, but in the majority of instances the disease remains limited in the hip-, shoulder-, or knee-joint.

Diagnosis.—Rheumatoid arthritis may be distinguished from gout by the polyarticular character of the disease, by its chronic and gradual development, by the absence of the violent attacks that are characteristic of acute gout, and by the lack of chalky deposits about the joints and in the tissues of the external ear and other parts of the body. Tubercular inflammation of the joints somewhat resembles rheumatoid arthritis, but it does not produce the characteristic deformities of that disease. Chronic rheumatism differs by the absence of characteristic articular changes and by the presence of conditions indicative of local inflammation. Sciatica may be differentiated by the distribution of pain along the course of the sciatic nerve and by the absence of rigidity and difficulty of movement from the hip-joint.

Prognosis.—Rheumatoid arthritis seldom proves fatal, unless complicated with other diseases of a dangerous tendency. When the joints have become deformed and permanently damaged by disease, they can never be restored to their original condition; but under judicious treatment, especially if undertaken at an early period, the disease may be arrested in certain cases and the swelling of the joints may be considerably reduced.

Treatment.—Debilitating measures and low diet exercise an unfavorable influence upon the course of rheumatoid arthritis. It is a disease that is characterized by debility and by an enfeebled nutrition, so that only in comparatively acute cases that are characterized by febrile symptoms is it expedient to place the patient in bed and to administer antiphlogistic remedies. Chronic cases may be allowed to take moderate exercise and to take as much wholesome food as the patient can relish and digest. Milk, eggs, and other forms of animal food should be liberally furnished for the diet of the patient, and such remedies as are found useful to aid digestion may be prescribed. Mustard, horseradish, and other cruciferous salads that contain sulphur are useful articles of diet. Unlike gout, the course of the disease is favorably modified by the use of alcoholic stimulants and beverages, taken in moderation with the meals. Cod-liver oil is an excellent nutrient, and should be administered in full doses after each meal. Various bitter tonics are beneficial chiefly as aids to digestion and stimulants of the appetite. The iodides are generally prescribed, but the iodide of iron is the most valuable member of this group. Iodide of potassium is particularly indicated in cases that are characterized by nocturnal pains. Arsenic is frequently beneficial, but its use is often followed by disappointment. By the French physicians great benefit has been ascribed to the use every other day of warm baths containing from fifteen grains to two drachms of the arseniate of sodium, together with three or four ounces of the bicarbonate of sodium. It is, however, probable that the good effects of these baths are due to their warmth and to the manipulation of the joints that accompanies such baths when administered by experienced hands. It is certain that careful massage of the limbs and movement of the affected joints while lying in a warm bath exercise a favorable influence in the restoration of the normal conditions of the affected extremities. It is the opinion of experienced observers that the undoubted benefit which is derived from these baths is due to their elevated temperature, to their long-continued repetition, and to the efficiency of the massage by which they are accompanied. So much depends upon the last method that much of the reputation of certain resorts is actually based upon the skill of the manipulators by whom the patients are attended while taking a course of baths.

Various other baths have been employed with more or less success. Turkish baths, Russian baths, hot baths in which the needle-shaped leaves of the pine have been steeped, vapors from infusions of juniper-berries and other terebinthinate plants, mud baths, sulphur baths, and salt-water baths, have all been employed with about the same results. Cold baths and sea-bathing are inadmissible. The patient should occupy dry, warm apartments remote from foggy and damp localities. The clothing should be warm and fully sufficient to meet the requirements of a rigorous season.

Acute exacerbations of the disease can be temporarily relieved by the use of salicylate of sodium, phenacetin, or salol, but in many cases it becomes necessary to resort to the use of opiates. Charcot recommends in such cases the administration of the carbonate of sodium to the amount of about an ounce, in divided doses, each day for a number of weeks. With this should be administered quinine in considerable doses. Guaiacum has been recommended, but is of little value except as a means of stimulating the circulation and promoting moderate perspiration.

Galvanism is recommended for the relief of pain and for the prevention of muscular atrophy and deformity. A large sponge electrode, representing the positive pole, should be placed upon the back of the neck or over the lumbar region, while the negative pole is connected with a dish of warm salt water in which the feet are placed. The duration of the treatment should not exceed ten or fifteen minutes every day for the first month, after which time the application may be made every other day or at longer intervals.

Of all the forms of medication that have been recommended, the most successful may be considered that by the daily use of warm baths, with the administration of the compound tincture of iodine or the syrup of the iodide of iron in increasing doses until the limit of toleration by the stomach has been reached.

GOUT.

BY HENRY M. LYMAN.

Definition.—A concise and accurate definition of gout is impossible. It is the most conspicuous manifestation of the arthritic diathesis. It is always a chronic disease, and when it is the result of hereditary causes it persists throughout the whole of life ; when acquired after birth it embitters the entire remainder of existence. Once acquired, the predisposition is permanent, and the more conspicuous outbreaks of the disease are to be considered as temporary exaggerations of a peculiar morbid constitutional condition in which the nutrition of the entire organism is retarded and perverted. Gout therefore belongs to that remarkable group of diseases which may exist either in the same individual or among members of the same family who have derived their morbid predispositions from a common ancestral source. Among these disorders are certain cutaneous diseases, such as eczema, impetigo, erythema, urticaria, and the allied mucous inflammations of the respiratory passages, together with chronic acid dyspepsia, congestion of the liver, lithiasis, hæmorrhoids, neuralgia, hemicrania, congestive headache, acute, chronic, and muscular rheumatism, asthma, obesity, diabetes, and polyuria. These disorders are all dependent upon a peculiar and ill-defined retardation of nutrition that constitutes the arthritic diathesis. It is impossible in the present state of our knowledge to explain with perfect satisfaction the nature of those errors of nutrition which determine the appearance of diabetes in one patient, obesity in another, and gout in a third. Their close relationship is, however, indicated by the common observation that they may be exhibited in succession by the same patient, and by their alternation with each other during the course of successive generations. The gouty patient often becomes the victim of obesity, while the diabetic subject may give birth to gouty progeny, which in its turn manifests now one and now another of the different diathetic diseases.

SYNONYMS.—Arthritis uratica, a. urica, a. vera ; Panarthritis urica ; Podagra ; Goutte ; Gicht ; Gutta.

LITHÆMIA.—The term lithæmia has been employed by many authors to denote the condition of the organism which is now recognized as constituting the arthritic diathesis. It is a condition that may be either inherited or acquired, and, once existing, it continues throughout the whole lifetime of the patient. It is dependent upon the same causes that produce the arthritic predisposition and that serve as a basis for the evolution of those diseases of nutrition which are characterized by an imperfect oxidation of the tissues. The prominent feature is an excess of uric acid in the system. It is accompanied by acid dyspepsia and the other disorders of digestion, circulation, and nervous function that characterize the arthritic diathesis. Chronic catarrhal gastro-enteritis is commonly observed, hæmorrhoids frequently exist, and there is a special predisposition to catarrhal affections of the respiratory mucous membranes. As a consequence of accumulation of uric acid in the blood and in the tissues the nervous structures of the body are subject to frequent paroxysms of irritation and excitement. Congestive headache, hemicrania, neuralgia, vertigo, tinnitus aurium, perversions of cutaneous sensibility, flushing of the face, burning sensations in the palms of the hands and in the soles of the feet, are common incidents in the experience of lithæmic subjects. The psychical functions are also liable to considerable disturbance through the alternations of cerebral excitement, with depression of spirits, irritability of temper, hypochondria, and a condition bordering upon melancholia. Cutaneous eruptions of an eczematous, urticarial, or lichenous character are not infrequent. The urine is high-colored, abundant, and acid, frequently depositing crystals of uric acid and the urates. In many instances the passage of urine is followed by scalding sensations and a feeling of soreness in the region of the kidneys, ureters, and bladder. All these symptoms are common incidents in the lifetime of persons whose habits of living lead to the local manifestations of gout, and they are not uncommon among temperate individuals who, by abstemiousness and a hygienic mode of life, never experience the fully-developed symptoms of gout, but would readily succumb to that disease after any departure from their ordinarily well-regulated mode of existence. For these reasons it seems unnecessary to perpetuate the term lithæmia, since the conditions that it denotes are identical with what have been described as the prominent features of the arthritic diathesis.

Etiology.—Gout is a disease that is rare in warm climates among people whose habits and education preclude the possibility of excessive indulgence in the use of food and drink. It is most prevalent among the luxurious inhabitants of large cities in the temperate zone, where civilization, wealth, and extravagance have been carried to the highest degree. The influence of climate is of less importance than the character of the dietary. It is the poverty and the consequent temperance of the dwellers in tropical countries that protect them from gout, and the disease is unknown in northern climates where for any reason similar abstemiousness from alcohol and animal food is enforced. The disease was well known among the debauched citizens of imperial Rome, and it has followed the transfer of luxury from Italian soil to the wealthy capitals of France, Germany, Holland, England, and America.

Gout is more frequent among men than among women. The reason for this preference is not fully understood, but it is probably connected with the greater disposition of the masculine sex to excessive exertion and self-indulgence in every direction.

Hereditary influences play an important part in the production of gout:

probably more than half the cases of the disease occur among the descendants of gouty ancestors. There is among authors no agreement with regard to the percentage of cases in which hereditary influences have been noted, some observers professing to trace them in every instance, while others can discover them in only 43 or 44 per cent. of the patients under review. The line of transmission is especially distinct upon the paternal side of the family. The predisposition is more frequently detected among the older and younger children, since they are more liable than the intermediate offspring to the manifestations of hereditary tendencies to the disease.

The local symptoms of acute gout are most frequently observed for the first time during the third decade of life, since the disease is most commonly encountered between the ages of twenty and forty-five years, when the sexual functions are most active. Debauchery, physical and intellectual fatigue, loss of sleep, and every form of excess or nervous excitement favor the development of the gouty paroxysm. The abuse of alcohol is not by itself likely to produce gout: it is when associated with sugar, dextrin, and other substances in the form of wine and beer that alcoholic beverages are most deleterious.

The intimate association between gout and other arthritic diseases is indicated by the ancestral prominence in gouty families of such diseases as obesity, rheumatism, diabetes, eczema, gravel, biliary lithiasis, hæmorrhoids, hemicrania, and neuralgia. A similar relationship between these disorders is illustrated by the fact that the local manifestations of gout are often preceded or succeeded by the same maladies that have just been enumerated. It is no exaggeration to affirm that the victims of gout are always ill, for despite the vigorous constitution and florid appearance that they often present to view, they are always liable to paroxysms of cutaneous, respiratory, digestive, urinary, or nervous disorder.

Symptoms.—The local manifestations of gout are either acute or chronic, but the predisposition, when it is hereditary, characterizes the whole period of life. There is, however, a wide difference between the mere arthritic diathesis and fully-developed gout. Children of gouty parents may possess a predisposition to gout by which they are rendered liable to many diseases which otherwise they would escape; but they may also experience non-arthritic forms of disease like other children who do not possess the predisposition to gouty disorder. In early childhood there is a notable tendency to the eruption of eczema and of impetigo, apparently as a consequence of the similarity of conditions that obtain during the infancy of individuals who on the one hand are predisposed to arthritic disorders, and on the other to scrofula and tuberculosis. The bond of resemblance between these two predispositions seems to lie in the fact of the retarded nutrition that is common to both.

During the period of childhood the possession of an arthritic predisposition is rendered conspicuous by the frequent occurrence of sudden, violent, and transient catarrhal affections of the respiratory mucous membranes. Coryza, tonsillitis, and bronchitis occur upon the slightest provocation, and sometimes are associated with urticaria in a manner that is suggestive of alternation between the disturbances of the cutaneous and the mucous surfaces of the body. At the age of puberty acute articular rheumatism sometimes occurs. Epistaxis is frequently witnessed, and headache often torments the growing boy or girl.

During the early adult life of the arthritic subject the urethral and genital mucous membranes are liable to severe and obstinate forms of inflammation, gonorrhœal or blennorrhagic, as the case may be. Herpetic eruptions about the prepuce are frequently experienced. Sometimes cystitis and epididymitis are developed through the upward extension of urethral inflammation.

At about the age of twenty-five years certain cutaneous eruptions are frequently manifested. Of these the most characteristic is eczema, developed symmetrically upon the lateral surfaces of the thumb, forefinger, and middle finger, and sometimes extending to the other fingers. It usually appears during the spring of the year, but vanishes during the summer months. Dry and scaly patches of eczema that itch frightfully are often developed over the sternum, about the ankles, in the armpits, and in the genito-crural folds of the skin, where, in cases complicated with corpulence, they present the appearance of erythema or intertrigo.

At the commencement of the fourth decade of life dyspepsia complicates the condition of the individual. Digestion is retarded, and the phenomena of gaseous eructation, gastro-intestinal flatulence, pyrosis, and an over-acid condition of the contents of the alimentary canal frequently annoy the patient. Constipation, headache, and the other symptoms of gastro-intestinal catarrh are the prominent features of this form of dyspepsia. The urine frequently deposits uric acid or urates. A tendency to hæmorrhoidal uneasiness now becomes manifest. Intense itching about the anus, the discharge of glairy mucus, sometimes tinged with blood, from the rectum, are not uncommon incidents, even though hæmorrhoidal tumors are not notably conspicuous. At the opposite extremity of the alimentary canal granular pharyngitis and suppurative tonsillitis are not uncommon. The transient catarrhal affections of the respiratory tract which characterize the period of infancy are now replaced by a chronic bronchitis. The naso-pharyngeal catarrh is liable to invade the Eustachian tubes, and leads to slowly progressive inflammation of the middle ear, resulting in more or less injury of the auditory function, and producing much of the deafness that exists during the later years of life. Paroxysms of hemicrania and congestive headache are frequently experienced. Boils sometimes occur in successive crops that serve to arouse a suspicion of diabetes, but the urine only contains urates and oxalates without sugar. Seborrhœa and pityriasis frequently attack the scalp, producing early loss of hair from the temples and the frontal region of the head.

With advancing years dyspeptic disorders become increasingly prominent. The liver is sometimes in a condition of tumefaction and excessive activity, but hepatic torpor is a common event. A coated tongue, foul breath, acid stomach, constipated bowels, headache, disinclination to intellectual effort, vertigo, nervous irritability, impatience, and ill-temper characterize a condition of ill-health that has been frequently designated by the term lithæmia. Sometimes slight swelling and tenderness of the articulations may be detected. Meat, starchy food, spices, coffee, tobacco, and alcohol aggravate all these symptoms. Sometimes a paroxysm of asthma or of renal colic or of intense gastralgia may serve to accentuate the other symptoms of the arthritic diathesis and to usher in the fully-developed attack of gout with its characteristic local manifestations.

It is usually at the close of a winter passed in gayety and dissipation that, after some unusual exposure to cold or fatigue of mind and body, preceded by symptoms of indigestion, languor, and constitutional disorder, the arthritic subject finds himself compelled by chilliness and fever to seek an uneasy couch at early bedtime. After a period of unrefreshing sleep he is awakened a little later than midnight by intense pain in the ball of the great toe, and to the agonizing condition thus initiated the phenomena of heat, redness, and swelling in the affected parts are soon added. Toward morning, however, suffering diminishes, and at last the patient sinks into an uneasy sleep as daylight appears. During the following day more or less suffering is experienced. The veins in the neighborhood of the swollen joint become distended, the pulse reaches 90 or 100 beats, and the temperature rises to 101° or 102° F. As evening approaches the temperature rises one or two degrees, and the patient is tormented by a return of the painful experiences of the previous night. During the succeeding day a remission is again experienced, and thus the disease progresses with daily remissions and nocturnal paroxysms of agony until about the fifth day, when all the symptoms begin to subside and convalescence is apparently established. Œdematous swelling of the tissues about the joint, having then reached its height, begins to diminish, and the epidermis wrinkles, cracks, and peels off, leaving an intensely itching surface. Suppuration of the joint never takes place, and uratic deposits only occur after the disease has become chronic. The urine is concentrated, high-colored, and deposits a sediment of urates.

During the first attack of acute gout the local symptoms are usually restricted to the great toe, but in subsequent attacks other joints are successively invaded.

The fever that accompanies the arthritic attack is characteristic. The conjunctivæ are injected; the countenance is pale; the tongue is broad, thickly furred, and moist, though it may become dry at the tip and along its central line. Thirst, loss of appetite, and constipation, with severe headache, are the usual accompaniments of the febrile movement. During the night mild delirium is sometimes experienced, and during the daytime there is a degree of irritability and ill-temper that is out of proportion with the severity of the local symptoms. In severe cases traces of albumin are sometimes discovered in the urine, but the symptom is transient and disappears as the febrile movement ceases. Perspiration does not occur until about the fourth day: it then appears, during the subsidence of the nocturnal paroxysm, in the form of a moderate transudation that is much less conspicuous than the reeking sweats that accompany rheumatism. At the height of the fever a certain amount of congestion may be sometimes detected at the base of one of the lungs, as if an attack of pneumonia were imminent.

The paroxysms of gouty fever appear to be connected with rapid destruction of uric acid in the organism. During the attack the serum of a blister that has been raised at a distance from the affected joint contains numerous crystals of uric acid, while serum drawn from the immediate vicinity of the articular swelling contains none: oxidation therefore appears to be more active at the seat of local disturbance. In this way may be explained the disappearance of uric acid from the blood and from the urine as the attack culminates and subsides. For this reason an attack of acute gout serves to clear the organism, and is followed by a prolonged period of freedom from the various morbid symptoms that preceded and ushered in the local manifestations of the disease.

When, however, by reason of repeated attacks, advancing age, or complicating disorders, the power of reaction is diminished, the patient no longer experiences the gouty paroxysm with typical intensity. The severity of the attacks is less pronounced; their onset is less abrupt; their subsidence is less complete; and the period of convalescence affords only an imperfect measure of relief from the general constitutional symptoms that are characteristic of the arthritic diathesis. In many instances a condition of chronic gout is established, and the patient no longer exhibits the phenomena of acute inflammatory disease. During the more or less frequent exacerbations of the malady very little fever is aroused; the joints are merely œdematous, without heat or redness, and such pain as is experienced is characterized by dull aching sensations instead of the sharp and lancinating agony that accompanies acute gout. The process of oxidation is no longer accelerated in the neighborhood of the affected joints: consequently uric acid, instead of being destroyed, is abundantly deposited in all the articular structures. These deposits form considerable masses around the capsule of the joint, constituting what are termed tophi. Thus infiltrated, the tissues undergo a secondary process of inflammation, and articular ankylosis is developed. The muscles by which an affected joint should be moved become atrophied as a consequence of reflex disturbance of their nutrition—a process that is most conspicuous in the extensor groups that lie between the joint and the parts nearer the body.

The relief that fails to be secured through the paroxysms of acute gout is sometimes obtained through the intervention of other acute inflammatory attacks which often appear to exhibit a truly substitutive character. In this way, various acute febrile diseases and inflammations, like tonsillitis, bronchitis, rheumatism, hæmorrhoids, epistaxis, and cutaneous eruptions, afford great relief from the constitutional symptoms of arthritism. The beneficial effects of such maladies are undoubtedly due to the increased activity in the process of oxidation and elimination that accompanies their course. This explanation is rendered still more probable by the alarming consequences that frequently follow the sudden arrest and suppression of such substitutive diseases

—a fact that was well known to the older physicians, who often gave warning of the dangers attendant upon retrocedent gout.

During the later stages of chronic gout the patient is confronted by two forms of danger—disease of the circulatory apparatus and disorganization of the kidneys. Progressive inflammation of the arterial walls may produce dry gangrene of the extremities or angina pectoris through impediment of the circulation of the blood; the heart may undergo fatty degeneration by reason of the imperfect nutrition of its walls through diminished blood-supply; interstitial nephritis may be in like manner excited, and be followed by all the characteristic symptoms of cardiac hypertrophy, bronchitis, pulmonary œdema, and uræmia.

An intimate connection between lead-poisoning and gout has been long recognized. It has been experimentally proved that the introduction of lead into the system interferes with the excretion of uric acid. Under the combined influence, therefore, of lead with alcohol, when imbibed in the form of beer, wine, or cider, the symptoms of gout are developed even among patients whose diet has not been extravagant and who have enjoyed abundant exercise in the open air. It is, however, rarely encountered before the development of the characteristic symptoms of lead-poisoning. Under such circumstances the gouty attack is not preceded by the well-marked symptoms that usher in uncomplicated gout, and the disorder is displayed in numerous joints at the same time, in this respect resembling the multiple incidence of chronic gout. The occurrence of tophi is less frequently observed, but visceral forms of gout and interstitial nephritis are commonly experienced. Death is in such cases often preceded by the phenomena of uræmia.

Pathology.—It is probable that during a first attack of gout no great modification of the affected joints remains as a permanent lesion; but when, after repeated attacks, an opportunity for dissection is afforded, the diarthrodial cartilages present a chalky appearance as if smeared with bismuth or lime. This change is due to an infiltration of the cartilage with urate of sodium. In advanced cases the articular surfaces are roughened and eroded. The ligaments, the articular cartilage, and the neighboring tendinous structures are also infiltrated with urates, chiefly the urate of sodium, with which is associated a small quantity of urate of calcium.

While it is true that joints which have exhibited before death no evidences of active disease may be found infiltrated with urates, the principal lesions are discovered in the joints that were most severely affected during the lifetime of the patient. It is, however, in the small joints of the upper extremities that the greatest amount of deformity, through the growth of tophi, is witnessed. The fingers become swelled and deformed; ulceration of the soft parts sometimes occurs, so that the periarticular concretions are laid bare. Old incrustations thus exposed often remain fixed, so that the patient can actually write his name upon a blackboard with his knuckles; but in many instances large quantities of semi-solid, pasty, and imperfectly crystallized urates are discharged through the ulcerated tissues. The large joints of the body are seldom thus invaded. Mineral infiltration occurs in those portions of the articular structures and in those joints that are least abundantly supplied with healthy nutriment through the medium of the circulation. The synovial membranes are sometimes also involved in the process of infiltration, and the synovial fluid is sometimes transformed into an acid paste that contains urates in considerable quantity. Occasionally the cavity of the joint contains a hæmorrhagic effusion.

The small joints are sometimes infiltrated to such a degree with calcareous urates that secondary inflammation results in complete ankylosis, which may occur without any marked deformity; but frequently the fingers and toes are greatly marred in form and position.

Besides the extensive concretions around the small joints, tophi exist in other parts of the body. Immediately under the skin these cutaneous tophi vary in size from the dimensions of a grain of sand to that of a large pea. They are usually found in the pinna of the ear along the border of the helix, upon the antihelix, and in the furrow which separates those two parts or upon the inner surface of the pinna. They are sometimes also discoverable upon the palmar surface of the fingers, in the eyelids and alæ of the nose, along the ulnar border of the forearm, over the inner surface of the tibia, and upon the corpora cavernosa of the penis.

Of all the internal organs of the body, the kidney is most frequently damaged by gout. The lesions are those which are characteristic of interstitial nephritis, but, in addition to the ordinary histological changes that characterize this form of chronic nephritis, the gouty kidney is marked by the presence of crystallized uric acid or urate of sodium in its substance. When uric acid predominates, it is found chiefly in the straight uriniferous tubules and in the pyramidal portions, calices, and pelvis of the organ, where it frequently excites the phenomena of pyelitis.

The deposit of urate of sodium chiefly occupies the connective tissue parallel to the uriniferous tubules in the pyramidal portion of the kidney. In the cortical zone and elsewhere evidences of necrosis involving circumscribed portions of the renal parenchyma are apparent. Cystic cavities containing urine thickened with urate of sodium are thus formed. Occasionally amyloid degeneration of the kidney is discovered, and sometimes the organ may exhibit a high degree of atrophy and contraction without any infiltration of uric acid or urate of sodium in its tissues.

The heart shares in the disorders that are manifested by the kidneys. As in other forms of interstitial nephritis, the left side of the organ becomes hypertrophied through the occurrence of a sclerotic process in the myocardium. Sometimes, however, the cavities of the heart become dilated and its muscular substance undergoes fatty degeneration. Occasionally a deposit of urates may be discovered upon the mitral or aortic valves. An atheromatous condition of the valves and walls of the arteries is more frequently observed. This atheromatous degeneration is often the cause of serious interference with the nutrition of the cardiac walls and of the brain and other organs: their blood-supply is reduced through partial obliteration of the vascular channels. Such atheromatous changes are originated at a much earlier period of life among arthritic subjects than among those who do not possess the arthritic diathesis.

Not only the arteries, but the veins and the capillaries, suffer from the evil influences of gout. The venous walls become relaxed, so that varicose veins and hæmorrhoids frequently add to the distress of the gouty patient. The capillary walls yield readily, so that hæmorrhagic effusions are not uncommon incidents in the course of gout.

Minute deposits of urate of sodium have been occasionally discovered in the cartilages and ligaments of the larynx and in the walls of the bronchi. Bronchitis is, however, frequently observed, though unaccompanied by such infiltrations. Emphysema, asthma, chronic bronchial catarrh, bronchial dilatation, and other pulmonary disorders are commonly experienced by gouty subjects.

The alimentary canal, the liver, and the intestines present the most conspicuous lesions. When gout is complicated with obesity the liver is usually affected by fatty degeneration. Cirrhosis is also a common event, though it may be usually referred to the habits of intemperance that have induced the occurrence of gout. The congestion and tumefaction of the liver which often exist are largely dependent upon the dilated and disordered condition of the stomach. The chronic gastric catarrh and consequent dyspepsia, though undoubtedly influenced by gouty conditions, are often referable to long-continued excesses in eating and drinking on the part of the patient. The catarrhal condition of the intestinal mucous membrane is usually dependent upon similar causes. Urates are only rarely detected in the alimentary canal.

The various cerebral phenomena, such as headache, vertigo, hæmorrhage, thrombosis, cerebral softening, etc., are dependent principally upon the condition of the vascular organs. Characteristic deposits of uric acid and urate of sodium are only occasionally to be found upon the meninges and other tissues of the brain and spinal cord. Such deposits have been, however, sometimes detected upon the external surface of the dura mater, compressing the roots of the spinal nerves and producing lightning pains in their course. The neurilemma of the peripheral nerves is sometimes invaded by a deposit of urates, and peripheral neuritis may be thus excited.

In the majority of patients suffering with either acute or chronic gout the presence of uric acid can be demonstrated in the serum of the blood. The familiar experiment of Garrod can be easily repeated, but it must not be forgotten that in rare instances articular deformity and the presence of tophi may indicate the existence of genuine gout without the possibility of detecting uric acid in the blood-serum. On the other hand, it has been shown that an excessive amount of uric acid may exist in the serum of patients who suffer with gravel, though never subjected to an attack of articular gout. In cirrhosis of the liver and in leucocythæmia the blood sometimes contains a much larger quantity of uric acid than in genuine cases of gout.

It was the opinion of Garrod that in all forms of gout, while the blood contained uric acid in excess, its amount in the urine was below the normal standard. French investigators, however, believe as a result of their observations that no such deficiency of uric acid in the urine can be determined. They also state that this excretion is considerably exaggerated both before and during attacks of the disease, and that its amount is only relatively diminished when the patient has become exhausted by chronic forms of gout.

The discharge of urea with the urine varies according to the diet of the patient; it is abundant when the food consists largely of animal matter, and and it is diminished during the attack when the patient rejects such nutriment. The phosphates vary with the urates. During the gouty attack the discharge is at first increased, but it gradually diminishes during it course. Before and after the attack the quantity of phosphates never falls below the normal figure, except in cases of cachexia and inability to digest the ordinary amount of food. For this reason the urine of persons who are predisposed to gout is habitually acid in consequence of an excessive presence of the acid phosphate of sodium. This salt holds in solution an excess of urate of sodium so long as the urine is warm: after cooling, the urates are deposited and are partly decomposed, yielding crystals of uric acid which are precipitated in the urinary sediment. These peculiarities of the urine characterize the majority of arthritic subjects, and to the over-acid condition of their blood should be referred the tendency to sclerotic changes in the kidneys, liver, and myocardium, together with a liability to the formation of renal and vesical calculi. It is probable that the same condition forms the underlying cause of those forms of sclerosis which so commonly invade the vascular and cerebro-spinal tissues among arthritic patients.

Treatment.—The prophylactic management of the gouty predisposition is of greater importance to the patient than mere remedial measures addressed to the acute attack of the disease. From earliest infancy the children of gouty parents should be subjected to a regimen that is calculated to obviate the tendency which has been inherited. The food should be properly proportioned, so that neither nitrogenous nor carbohydrate elements may be in excess. A mixed diet that is not too abundant is to be preferred. Milk and water should form the only beverages of the growing child. Tea, coffee, and, above all, alcoholic stimulants, should be forbidden. Ale, porter, beer and cider are especially deleterious. Among gouty subjects a single glass of champagne is sometimes sufficient to induce an attack of gout. If alcoholic stimulants must be permitted, the choice should fall upon distilled spirits without sugar or upon white wine or old Bordeaux wine, but champagne and rich sweet wines, especially those that contain tannin in considerable quantity, must be avoided.

Since imperfect oxidation of the tissues is one of the principal characteristics of the arthritic diathesis, it is necessary that abundant exercise in the open air be taken in order to promote active respiration and the assimilation of oxygen. Elderly persons who lead a sedentary life should be encouraged to active habits. Horseback exercise, mountain-climbing, boxing, fencing, and other forms of gymnastic exercise are useful during early life, but if the organs of circulation have become diseased, and when age precludes the possibility of great activity, passive exercises, like massage and the Swedish movements, are of great value. All forms of fatigue must be avoided, since an acute attack of gout is often induced by a neglect of this precaution.

Warm clothing must always be provided ; dampness and cold are not tolerated by gouty subjects. The skin should be fortified by dry rubbing, and must be protected with flannels. Elderly and feeble patients should seek a warm and dry climate during the winter months.

It is undesirable to attempt abortive treatment of an attack of acute gout. Bloodletting, leeching, and the use of the numerous specifics that are widely advertised cause more harm than good. The remedial and depurative nature of the access of acute gout is thoroughly understood, and the patient should be instructed accordingly. He must remain in bed or in a recumbent position for several days. The affected limb should be firmly supported upon a pillow, and be covered with a light frame for the support of the bed-clothes. The inflamed joint may be kept moist with hot whiskey and water applied upon absorbent cotton, which may be also sprinkled with tincture of opium and tincture of belladonna. Duckworth recommends painting of the joint with oleic acid in an ounce of which have been dissolved three grains of atropine and fifteen grains of hydrochlorate of morphia. Iodoform, oil of peppermint, a solution of cocaine, and flexible collodion are also recommended.

At the outset of the attack the bowels should be cleared out with calomel and colocynth. Two grains of calomel at night, followed by one or two Seidlitz powders the next morning, are usually sufficiently aperient.

When the local manifestations of the disease are fully displayed in the affected joint, the administration of colchicum may be commenced. The prescription that is such a favorite in St. Bartholomew's Hospital, London, forms an excellent vehicle for the drug :

℞. Magnesii carbonatis,	gr. x ;
Tinct. colchici sem.,	♏xx ;
Aq. menth. virid.,	ad f℥j.

A half drachm of the wine of colchicum in a wine-glass of the mistura sennæ composita may be given every morning. Veratrine has also been employed externally and internally, though it is less efficient than colchicum. It is supposed to be the active agent in Laville's liquor. The use of colchicum should never be carried to the extent of producing irritation of the stomach or purging of the bowels. During the intervals between gouty attacks the drug may be administered in small doses for a long period of time with great advantage, especially when combined with tonic doses of quinine. In many cases an attack of gout may be successfully treated with the various salts of potassium to the exclusion of colchicum. The French physicians favor the postponement of colchicum until the later period of the attack; but in old and frequently recurring cases an early exhibition of the remedy is desirable.

Salicylic acid and the salicylates have been highly recommended in the treatment of gout, and they are very efficient remedies when given in large doses. They are, however, less satisfactory than colchicum for the relief of the local manifestations of the disease. They are especially useful during the subsidence of the attack and in cases that are complicated with rheumatism, and in the headache that is dependent upon an excess of uric acid in the blood. The existence of renal disease or cardiac debility should be regarded as contra-indicating their use. Phenacetin, acetanilid, and antipyrine are useful during the febrile stage of the attack, and they also diminish pain to a certain extent.

Of the alkaline salts that are useful in gout, the citrate, acetate, and bicarbonate of potassium, in doses of fifteen to thirty grains every two hours, dissolved in a large quantity of water, are the most useful. Lithia-water and the salts of lithium are very valuable, especially in the chronic forms of gout. Phosphate of sodium is a desirable laxative, though sodium salts cannot always be tolerated by gouty patients. During the acute stage the salts of potassium are the most useful of alkaline remedies, but during the intervals between attacks sodium salts and mineral waters which, like Vichy water, contain sodium, are usually better tolerated than the compounds of potassium. Whenever the alkaline treatment tends to cause debility, it should be reinforced with tonic doses of quinine or the various tinctures and elixirs of Peruvian bark.

The severe pain that is experienced during acute gout may be relieved by the cautious administration of chloral hydrate. The administration of opiates and hypodermic injections of morphine must not be permitted. Vomiting may be relieved by sucking ice. Hiccough may be suppressed by giving a few drops of chloroform dissolved in ice-water; mustard plasters and hot poultices should be applied to the epigastrium when there is complaint of pain in that region.

The management of chronic gout requires careful observance of ordinary hygienic rules. The diet must be properly assorted, avoiding a preponderance of starchy and saccharine elements as well as an excessive indulgence in animal food and malt liquors. An occasional dose of blue pill, followed by a Seidlitz powder in the morning, is of great value as a means of obviating hepatic engorgement. Vigorous and rubicund patients who make blood readily may be advised to drink the Saratoga, Carlsbad, Hunyadi Jànos, Friedrichshall, Püllna, Rubinat, or Crab Orchard waters. Whenever anything approaching an acute attack is manifested small doses of colchicum may be administered. Iodide of potassium is useful when the periarticular tissues are considerably involved. The elixirs and tinctures of Peruvian bark, and quinine with nux vomica or strychnine in tonic doses, are exceedingly beneficial in atonic forms of chronic gout. Guaiac is especially useful when the circulation is feeble. It may be given in the form of the ammoniated tincture, or in an effervescing powder containing five grains of powdered gum guaiac with twenty grains of bicarbonate of potassium in four ounces of lemonade. It should be taken often enough to produce a gently laxative effect. In advanced cases of the disease that are complicated with albuminuria and anæmia the potassio-tartrate of iron may be administered cautiously, with a saline laxative to prevent headache and constipation. The chalybeate waters in moderate doses are sometimes beneficial in such cases, but in many instances iron cannot be tolerated by the gouty patient. In such a dilemma small doses of manganese have been recommended, but arsenic is the most generally useful tonic; it may be given in the form of Fowler's solution, with quinine, nux vomica, and other tonics.

The occurrence of retrocedent gout demands active counter-irritation for the purpose of re-exciting inflammation in the articulations that were primarily affected. Hot mustard foot-baths and mustard poultices applied to the epigastrium are exceedingly useful. A mercurial cathartic should be administered, and the excessive flatulence which is often occasioned by retrocedence of the gouty attack may be relieved by the administration of Hoffmann's anodyne, tincture of camphor, or spirit of peppermint. Plethoric patients with symptoms of cerebral congestion are sometimes relieved by venesection.

In the minor and irregular manifestations of the arthritic diathesis commonly described as lithæmia the diet should be regulated in accordance with the principles that have been already enunciated. Potatoes and other starchy vegetables should be avoided, as well as saccharine and fatty substances. Lettuce, celery, water-cresses, cucumbers, onions, horse-radish, cabbage, tomatoes, oranges, lemons, and other fruits that contain an abundance of acid salts of potassium may be allowed, unless there be an intolerance of the vegetable acids. Lean meat, eggs, fish, and skimmed milk, with bread in moderation, should constitute the principal portion of the food. Alcoholic beverages, coffee, tea, and tobacco must be prohibited. When the contents of the stomach are excessively acid through fermentation, nitric acid in five-drop doses largely diluted should be administered before each meal. Alkaline salts are principally valuable in connection with excessive acidity of the intestinal canal, accompanied by fermentation and flatulence in the bowels. The salts of sodium should not be administered for any considerable length of time, since they promote the formation of urate of sodium, which is insoluble. The salts of lithium and of potassium, on the contrary, exercise no such influence. They should be given about three hours after each meal, so that they may pass into the intestines near the close of the gastric digestion. The great value of the "neutralizing cordial," which contains the bicarbonate of sodium and of potassium, is largely due to its action in obviating intestinal acidity. Vichy water, Londonderry lithia-water, and other alkaline mineral waters are similarly beneficial. When constipation is a prominent symptom, phosphate of sodium may be given in daily doses for many months, and it may be profitably associated with podophyllin, euonymin, aloin, and rhubarb when a cholagogue effect is desirable.

The disorders of the nervous system that are dependent upon the arthritic diathesis demand the same general treatment that is appropriate in cases of chronic gout. Insomnia is frequently dependent upon the gouty predisposition. Various forms of neuralgia and neuritis are similarly developed; headache and hemicrania not unfrequently torment the arthritic patient. Vertigo and hysteria that own a gouty relationship are not uncommon. In certain cases muscular spasms, especially in the calves of the legs, may be observed, and a similar tendency is frequently indicated by fibrillary twitchings in the facial muscles or in other parts of the body. Epilepsy when occurring in arthritic subjects is considerably aggravated, and may be relieved by attention to the underlying diathesis. In certain rare instances local paralysis has been observed as a consequence of peripheral neuritis of a gouty character.

A tendency to bronchitis and pneumonia has been already mentioned. Asthma often occurs either in association with neurotic disturbances, bronchial inflammation, or as a consequence of retrocedent gout. In all such cases treatment of the local condition must be subordinated to the general management of the constitutional disorder.

It is through the circulatory apparatus that many of the evil consequences of arthritism find their expression. During early and middle life palpitation, irregular and intermittent action of the heart, valvular disease, and angina pectoris are frequently observed. In later life arterio-sclerosis and atheroma are the cause of imperfect nutrition and final destruction of function in the heart itself, in the brain, spinal cord, and other important organs of the body. Phlebitis and consequent thrombosis are sometimes developed, especially in the lower extremities. In the course of the alimentary canal neuralgia involving the tongue, and the peculiar patches constituting leucoplasia of the tongue, are sometimes observed. Tonsillitis and parotitis are often dependent upon the arthritic predisposition. A tendency to gastro-intestinal catarrhal affections is a matter of common observation, and is the cause of much ill-health among arthritic patients. Renal inflammation and renal lithiasis have already been sufficiently noticed. In connection with the external urinary organs, balanitis, pruritus of the vulva, herpes præputialis, and orchitis are not uncommon incidents. Persistent painful priapism is occasionally experienced; it is relieved by the administration of iodide of potassium with alkalies and other antiarthritic remedies. Gouty urethritis and cystitis are not uncommon complications of a tendency to lithiasis. Among female patients painful conditions of the ovaries and pelvic organs are exceedingly common, and are intractable so long as their relation with the arthritic diathesis remains unrecognized.

RHEUMATISM.

By HENRY M. LYMAN.

Long confounded with gout, rheumatism is now universally recognized as a general disease affecting the entire body, but accompanied by local manifestations involving chiefly the fibrous tissues of the articulations and locomotive apparatus, yet frequently invading any or all of the structures of the body, and in its acute forms exhibiting many of the characteristics of circumscribed inflammation, Its subacute, chronic, and visceral forms, however, may be developed without appreciable swelling or elevation of temperature. So numerous and so various are the varieties of the disease that no general description can be formulated as a definition of the term "rheumatism." The ignorance that yet prevails regarding the ultimate cause of the disease renders it impossible to indicate precisely the number and nature of its different manifestations. It is therefore frequently confounded in certain of its forms with clinically similar exhibitions of gout, and with the secondary consequences that sometimes follow septic poisoning, gonorrhœal inflammation, and other toxæmic disorders. The fact that the chronic forms of gout and of rheumatism present many points of clinical resemblance, and also the fact that many of the phenomena of gout and rheumatism may concur in the same patient, tend still further to confuse the diagnosis in some of the obscure forms of these diseases. It is in the acute forms of rheumatism that the possibility of a differential diagnosis is rendered easy. It is in this form, therefore, that the typical picture of rheumatism can be most clearly recognized.

It will be found convenient to arrange the varieties of rheumatism into three classes: 1, Articular Rheumatism; 2, Rheumatism of other organs, either external or internal, constituting what may be termed Abarticular Rheumatism; 3, General, Diffuse, Non-circumscribed Rheumatism. Besides these three classes, a fourth may be established for the inclusion of Secondary or Pseudo-rheumatic diseases, such as gonorrhœal, puerperal, menstrual, scarlatinal, syphilitic, dysenteric, and malarial arthropathies.

ACUTE ARTICULAR RHEUMATISM.

Etiology.—Acute articular rheumatism is one of the most common of diseases. In the hospitals of Paris and of Vienna it constitutes from 3 to 5 per cent. of all the admissions. It is a disease that probably affects alike all races of men.

The influence of climate upon the occurrence of acute articular rheumatism has not been subjected to the complete test of statistical research upon a large scale. But it is generally believed that the disease prevails less frequently in arctic and tropical regions than in the temperate zones of the earth. Uniformly cold and uniformly hot weather seem to be unfavorable to the development of the disease. It prevails most frequently where the weather is variable and damp.

The influence of temperature appears to be less uniform than the influence of exposure to wet and cold. According to the French observers, the greatest mortality of rheumatism in Paris is observed during the summer months, while in Copenhagen and in the north of Europe the greatest prevalence of the disease is during the winter months.

The elevation of the habitat above the sea-level exerts some influence upon the prevalence of rheumatism, yet less than is observed in many other diseases. While the majority of cases occur in low and damp localities, many instances of the disease are encountered upon elevated plateaus. By far the greater number, however, are found in cold, bleak, and damp regions. Lange of Copenhagen has expressed the opinion that the prevalence of acute rheumatism is subject to epidemic aggravations which are not dependent upon ordinary meteorological conditions. No explanation of these occasional epidemics has yet been given.

In addition to the epidemic variation of acute rheumatism, it has been noticed that the disease appears with variable severity and variable modes of manifestation at different periods of time. Thus, the occurrence of endocarditis or of pericarditis may be frequently observed during one season, while at other times it rarely occurs. During certain epidemics the disease may be exceedingly modified, while in other years it may be characterized by great severity and considerable fatality.

Certain observers have noticed a tendency to the local prevalence of rheumatism in particular houses, but others, equally competent, have failed to note any such coincidences. The influence of dampness and lack of drainage in the soil underneath the dwelling is probably the principal cause of such local prevalence of rheumatism.

The question of an infective cause for acute rheumatism has not been decided. Many of the symptoms of the disease and its mode of prevalence seem to be indicative of an infective cause. There is, however, no satisfactory evidence of the transmissibility of the disease from one person to another, nor has any infective micro-organism been discovered that can excite symptoms of rheumatism. It is said that women suffering with acute rheumatism have given birth to children who almost immediately exhibited the symptoms of the same disease, but such observations are not yet sufficiently numerous nor sufficiently accurate to be employed as positive evidences in favor of the infective hypothesis.

Acute articular rheumatism occurs at all ages, but it is less frequent during childhood than in adult life. Of 8631 cases collected by Besnier, there were only 301 children. In early infancy the majority of so-called cases of rheumatism are of a syphilitic or septic character. Occurring occasionally between the fifth and fifteenth years, it is more commonly experienced after the twentieth year, reaches its maximum of frequency between the thirtieth and fortieth, becomes less frequent after the fifty-fifth year, and during the remainder of life is usually replaced by subacute forms of the disease.

The incidence of acute rheumatism upon the two sexes is about equal.

Hereditary causes exert an important influence upon the occurrence of articular rheumatism. It is of course impossible to determine exactly the proportion of cases in which such conditions are operative, but, so far as statistics have been collected, it seems probable that from one-quarter to one-third of those afflicted are the descendants of rheumatic ancestors.

Whether rheumatism is or is not to be considered a manifestation of the arthritic diathesis depends upon the determination of its ultimate cause. So long as it is in any way confounded with gout, there will be a strong inclination to consider it as a diathetic disease based upon a variety of the arthritic diathesis. Many of the traits that have been considered indicative of a rheumatic constitution and temperament are simply expressions of a scrofulous or gouty predisposition. It is nevertheless a fact that while articular rheumatism may attack subjects of the most varied constitution and temperament, it is most frequently experienced by delicately organized patients who perspire inordinately, and are thereby rendered liable to sudden and excessive refrigeration of the body.

The occupation and social life of individuals exert less influence upon the occurrence of articular rheumatism than might be expected. It is only when they entail exposure to the exciting causes of the disease that they exert any marked influence. Exposure to dampness, associated with frequent fluctuations of temperature, forms one of the most efficient causes of the disease; consequently it is commonly observed among laborers, cooks, blacksmiths, factory-hands, and domestic servants. While it is by no means restricted to the poorer classes, it is far more common among them than among the wealthy, who are more frequently subject to gout as a consequence of luxurious living and excessive indulgence in eating and drinking.

Among the most common of the exciting causes of acute articular rheumatism must be mentioned sudden refrigeration by exposure to dampness and cold. Dry cold exercises comparatively little influence. It has been mentioned by Arctic explorers that during the long and icy winter rheumatism was unknown, but with the return of thawing weather in the spring and summer the disease reappeared. Mere refrigeration, however, is insufficient to excite the disease. Many persons who have undergone such exposure never experience rheumatism, though they may yield to other inflammatory disorders. A third unknown agent is necessary for the production of rheumatism. If the hypothesis of infection should ever be established, it would become easy to understand the manner in which refrigeration operates, by comparison of the invasion of rheumatism with that of chicken cholera in fowls which have been subjected to the action of cold and moisture by immersion in cold water.

Excessive fatigue, especially when accompanied by inordinate use of the limbs and consequent temporary deterioration of the articular structures, is an active exciting cause of rheumatism among those subjects who are predisposed to its occurrence. It has long been observed that the incidence of acute rheumatism involves those articulations and those organs of the body that have been exhausted by excessive activity before the attack.

Injuries and surgical diseases not unfrequently serve to excite an attack of rheumatism in the affected parts or in their immediate neighborhood; thus a sprain, a blow, a fall, or a localized inflammation may be followed by an attack of acute articular rheumatism in immediate proximity with the injured part. In such cases the latent predisposition is unmasked by the depressing influence of the traumatism or surgical disease. It has also been observed that injuries involving rheumatic joints tend to cause the establishment of chronic rheumatism as a sequel of the acute attack.

The sudden suppression of perspiration or other habitual evacuations, such as epistaxis, hæmorrhoidal hæmorrhage, and the menstrual flow, has been assigned as one of the exciting causes of the disease. While it is true that excess in eating and drinking are usually predisposing causes of gout rather than of rheumatism, it is also true that alcoholic excesses may by their depressing influence upon the general health favor the occurrence of rheumatism, and there is considerable evidence to show that they exercise a notable influence in determining the localization of rheumatism in the internal organs, especially upon the cerebral membranes. It is probable that when rheumatism attacks the viscera of alcoholic subjects its effects are more permanent and severe than they would have been otherwise.

Pathology.—Rheumatic inflammation is limited to no particular tissue, but involves all the tissues and structures that unite in the constitution of an articulation. The subcutaneous connective tissue is infiltrated with products of inflammation, and exhibits ecchymotic points. The intermuscular connective tissue exhibits similar alterations, and is occasionally the seat of actual suppuration. The sheaths of the muscles and of the tendons are reddened, injected, and filled with serous liquid, sometimes containing pus-cells and flakes of fibrin: the tendons themselves may remain healthy, but are sometimes considerably inflamed. Occasionally the palmar aponeurosis undergoes inflammation and permanent contraction, even though the joints of the extremities are not affected.

The synovial membrane and the synovial fringes exhibit great vascularity, thickening, and varicosity of the vessels. Microscopical examination reveals extensive cellular proliferation, especially involving the synovial fringes. The articular cavity becomes distended by an increase of the synovial fluid, which sometimes yields an acid reaction instead of its normal alkaline quality. Sometimes the fluid is stained with blood as a consequence of hæmorrhage into the cavity of the joint. Occasionally, in certain cases involving a single joint or only a few of the articulations, the cavity contains pus, but ordinarily the exudation differs very little from the normal synovial fluid. Sometimes, however, numerous large compound granular corpuscles are present, giving a turbid appearance to the liquid. Pus-corpuscles are usually present in small numbers, but it is very seldom that they are sufficiently numerous to constitute a purulent exudation.

The articular cartilages appear to be deprived of their normal polished surface. The cartilage-cells increase in number by segmentation, a process that is most conspicuous in detached islets scattered here and there throughout the cartilage. In the deeper layers of the diarthrodial cartilage the capsules that have been distended by multiplication of their cellular contents are arranged in rows perpendicular to the articular surface, while the superficial capsules lie parallel with that surface. At the same time, the interstitial substance of the cartilage splits up in lines parallel with the rows of capsules, giving to the cartilaginous substance a somewhat velvety appearance. When ulceration takes place the interstitial substance becomes liquefied, and the cartilaginous cells are liberated and discharged into the cavity of the joint.

When the disease progresses for a considerable length of time the bone-marrow near the extremities of the long bones exhibits an increase of vascularity with active proliferation of its cells. The periosteum also exhibits a similar increase of vascularity.

The pathological changes that have been thus enumerated are developed with great rapidity, and they may exist even though the external appearance of the affected joint gives little reason to suspect such extensive alterations of structure. It is, however, evident, from the rapidity and the completeness with which recovery usually takes place after acute articular rheumatism, that the processes of repair may be effected as rapidly as the process of disintegration. It should not be forgotten, however, that the lesions of the synovial capsule of the joint disappear more quickly than those of the cartilaginous structures of the articulation ; consequently, the subsidence of swelling and the disappearance of the external signs of inflammation should not be accepted as evidence of complete recovery in the cartilaginous and osseous tissues. The affected joint should therefore be protected from all unnecessary use for a considerable time after the subsidence of acute symptoms.

The blood exhibits an increase of fibrin, which may reach two or three times the normal quantity. When drawn from the veins this increase of fibrin causes the clot to assume that peculiar cupped appearance which the older physicians considered so important a sign of inflammation. It is to this increase of the elements of fibrin that is to be referred the formation of clots in the heart and in the arteries, together with vegetations upon the cardiac valves. The red blood-corpuscles are rapidly diminished in number. In cases of cerebral rheumatism characterized by high temperature the blood is black and incoagulable, like the blood in cases of septicæmia. Sometimes the capillary walls also undergo morbid changes that permit the transudation of the coloring matter of the blood-corpuscles or the red corpuscles themselves, thus constituting hæmorrhagic rheumatism.

Uric acid does not exist in any abnormal quantity in the blood of rheumatic patients, nor is the quantity of urea increased. The serum of the blood preserves its alkaline quality. The fats, cholesterin, and extractive matters are increased in quantity.

The urine is generally high colored and very acid, depositing as it cools a brick-dust sediment that contains amorphous urates and occasionally crystals of uric acid. The specific gravity is high, since the urine is scanty and concentrated by copious perspiration, but during the remission of fever and after the commencement of convalescence the urine assumes its normal quantity and appearance. Albumin is not usually present unless the patient becomes cachectic; a small quantity of albumin may then appear in the urine, but it disappears with the return of health.

The secretions of the liver and of the intestines are diminished during an attack of acute rheumatism, and constipation usually exists.

The tendency to excessive perspiration that is characteristic of a predisposition to rheumatism becomes greatly exaggerated during an acute attack of the disease: this excessive perspiration is one of the most constant and characteristic symptoms. The reaction of the fluid is nearly neutral if the surface of the skin be kept perfectly clean. It exhibits a greater degree of acidity when the transudation is less abundant; alkaline perspiration is occasionally observed. The apparent excess of acidity upon the surface of the skin is the result of decomposition of sebaceous matter and epithelial débris that have been allowed to accumulate upon the cutaneous surface. Consequently, it follows that the perspiration which accompanies acute rheumatism is a morbid phenomenon rather than a critical or beneficial evacuation.

Symptoms.—The course of acute rheumatism is subject to such wide variations of intensity, duration, and character of the phenomena that it is impossible to present any clearly defined symptomatic picture of the disease. Sometimes the attack commences suddenly in the midst of apparent health, but more frequently the onset of the disease is preceded by a condition of debility or of nervous disturbance. Sometimes it is apparently excited by a debauch or by an injury.

In the majority of instances the attack of rheumatic fever is preceded for a variable period of time by sensations of stiffness in the joints or by various vague disturbances of health which may be the common inflammatory consequences of ordinary exposure to cold. Articular pains and sensations of fatigue are experienced. The slightest exertion arouses perspiration, accompanied by a feeling of chilliness. Under such conditions a moderate exposure to chill air, or a hot bath followed by a cold douche, is often sufficient to precipitate the attack. Occasionally the onset of the disease counterfeits the paroxysm of gout, or the attack may assume the form of sciatica or other ordinary neuralgia. Sometimes certain muscles become swelled and painful before the joints are invaded. Occasionally the attack is localized in some one of the internal organs or tissues of the body before the articulations of the extremities are invaded. In certain rare cases the lower limbs are simultaneously crippled by a sudden attack of acute rheumatism, which may be mistaken at first for a paraplegic affection.

The local manifestations of rheumatism are seldom preceded by any well-defined febrile movement. Fever accompanies or follows their development, being a symptomatic rather than an essential fever. When it seems to precede the articular disorder, it is usually associated with some internal or easily neglected manifestation of the disease. In such circumstances the fever may be symptomatic of a rheumatic tonsillitis or inflammation of the respiratory tract or of the heart itself. Occasionally, however, the fever may appear before it is possible to discover any local evidences of rheumatism.

The invasion of one of the joints is indicated by more or less swelling, redness, heat, and pain. Elevation of temperature can be easily distinguished by applying the palm of the hand to the affected part, even though redness and swelling are not apparent. These symptoms are most conspicuous in the joints which are but thinly covered by the skin and subcutaneous tissues. In the shoulder and in the hip redness and swelling are seldom present, while at the knee, the ankle, and the wrist they are very evident. When the inflammation is severe the skin over the affected joint exhibits an erythematous color, which disappears under the pressure of the finger, but reappears with great rapidity when that pressure is removed. In many instances there is considerable œdema about those joints which are surrounded by loose connective tissue, as on the wrist, upon the back of the hands, and on the ankles. Under such circumstances the amount of redness and swelling often suggests the idea of erysipelas or other non-rheumatic inflammation. It is, however, closely restricted within the limits of the periarticular tissues and the sheaths of the tendons. In acute cases the superficial veins are dilated, and the arteries beat powerfully within the area of inflammation; but in subacute forms of the disease, though there be increased heat and swelling about the joint, the skin remains white, tense, and smooth by reason of the œdematous condition of the subcutaneous tissues.

The phenomenon of swelling is dependent not only upon periarticular œdema, but upon effusion within the articular cavity. It is in the knee especially that the effects of such distension are most conspicuous.

During the course of articular rheumatism movement of the joint is frequently accompanied by audible sounds or by creaking like that of new leather, which may be felt when the hand is placed upon the affected joint. Sometimes it is possible to discover in the skin, in the subcutaneous connective tissue, in the tendinous sheaths, and upon the periosteum certain nodular masses which can be detected by palpation, though they are sometimes visible to the eye. These little tumors can be sometimes discovered under the scalp or on any other part of the body, constituting little movable masses varying in size from a small pea to a bird's egg. They are not painful to the touch, and their appearance and disappearance succeed each other with great rapidity.

The position of the affected limbs varies according to the number of joints that are involved and the amount of inflammation that is present. The upper extremities usually assume an attitude of semiflexion and pronation. The fingers are generally extended. When numerous joints are simultaneously involved the patient lies motionless; the lower limbs are semiflexed and partially abducted; and every effort to change the position that is thus spontaneously assumed is attended with the greatest suffering.

The pain that is experienced has its seat in the subendothelial network of nerves and blood-vessels. It is often increased at night, and either ceases altogether or is diminished in severity when effusion takes place into the articular tissues and cavities. It is aggravated by pressure and by movement, so that the patient in polyarticular rheumatism is compelled to remain as motionless as if utterly paralyzed.

The joints of the lower extremities are invaded more frequently than those of the upper. The knee and the ankle are most frequently attacked, and it is in the wrist and the knee that the disease lingers the longest. The disease is usually symmetrically developed upon the two sides of the body, attacking the larger joints either simultaneously or in succession. The smaller joints of the extremities are less frequently involved. As a general rule, the larger the number of joints that are invaded, the less the persistence of the local manifestations in any particular joint. The mere disappearance of swelling and pain must not, however, be accepted as evidence of complete restoration in the tissues of the affected articulation: a certain amount of morbid change endures throughout the entire attack. When uninfluenced by treatment the average duration of painful swelling varies from four to eight days, though in many instances it is much longer. The local symptoms are liable to alternate exacerbations and remissions by which the total duration of the attack may be prolonged for several weeks. The cause of these oscillations is unknown. Abortive attacks are sometimes witnessed, in which a violent invasion of the disease is succeeded by a spontaneous disappearance of all acute symptoms and a rapid recovery. In certain cases, however, complete and rapid disappearance of the external phenomena of the disease is speedily followed by the development of the alarming symptoms of cerebral rheumatism.

In the majority of cases the local lesions completely subside, but occasionally the affected joints become permanently crippled and deformed by persistent changes in the fibrous capsules of the joints. Such permanent injury is usually caused, not by the rheumatism itself, but by complications that are dependent upon a scrofulous diathesis.

The febrile movement that accompanies rheumatism lacks many of the characteristics of ordinary inflammatory fever. The surface of the body remains pale, notwithstanding copious perspiration and elevation of temperature. In many cases the appetite and digestion are but slightly affected; delirium is rare; headache is not severe, though sleep is greatly disturbed by nocturnal pain. The mind remains unaffected except in cases of cerebral rheumatism. Hæmorrhages sometimes occur during the course of acute articular rheumatism, but in the majority of cases they are dependent upon rheumatic congestion of the lungs, upon changes in the blood, or upon embolism or thrombosis in different organs of the body. Epistaxis, however, is frequently experienced, but is usually without special importance in relation to the course of the disease.

The frequency of the pulse in moderate forms of rheumatism is but slightly increased: in severe cases it may rise to 100 beats or more; a pulse-beat of 120 or upward is usually indicative of complications.

Nothing can be more variable than the temperature in a case of acute rheumatism. In ordinary cases it rises but little above 100° F.: a temperature of 104° or 105° F. that persists for a brief period only is not uncommon nor alarming in severe cases, but persistent and excessive elevation of the temperature constitutes a dangerous symptom, especially when it is associated with cerebral symptoms. The daily oscillations of the thermometric curve are irregular and of unusual amplitude. The period of defervescence is usually characterized by a gradual and rather irregular fall of temperature. Critical subsidence is not commonly witnessed.

The copious perspiration that accompanies rheumatism is an evidence of profound disturbance of the nervous system. It is a morbid phenomenon, and is not to be considered as a critical or beneficial evacuation—a fact that is illustrated by the good effects of remedies, like atropine, which serve to moderate the amount of transudation. The miliary and sudaminous eruptions which accompany perspiration have no specific relation to the rheumatic attack, since they are commonly observed whenever the skin is irritated by excessive perspiration and lack of cleanliness. They are usually aggravated by a high temperature and by excessive generalization of the disease; their appearance is not without prognostic significance.

The **course** of the disease is liable to great irregularity and diversity. Sometimes a mild invasion is succeeded by intense aggravation of all the symptoms during the fastigium of the fever; the reverse of this order is also sometimes observed. So much depends upon the localization or the general diffusion of the manifestations of the disease, upon its restriction within the limits of the articulations, or its extension to the internal organs of the body, that no uniformity can be anticipated in the evolution of the various symptoms which constitute and complete the attack. In addition to the repeated exacerbations and paroxysms of articular disorder, genuine relapses of the disease are not uncommon during the period of convalescence. Such renewal of the symptoms after their apparent subsidence is not uncommon as a consequence of premature suspension of treatment when salicylic acid and its compounds have been employed at an early period in the course of the disease. In certain cases a recurrence of the attack may be experienced many times in succession, even though the intervals are characterized by complete restoration of the health.

The **duration** of the rheumatic attack is exceedingly variable. In many cases, despite the disappearance of acute symptoms, the patient remains for a long time pale and debilitated. The average duration of a moderately severe attack is from two to four weeks. Ordinary cases persist from three to six weeks, but severe forms of the disease may last for three months.

In the great majority of cases acute articular rheumatism terminates in complete recovery ; death, however, occasionally results as a direct consequence of the attack. When, after a distinctly acute articular course, the disease degenerates into an indefinitely prolonged arthritis, the local disorder is usually of a scrofulous character rather than a form of chronic rheumatism. Such persistence of arthritic disease is usually witnessed when only a single joint was originally involved.

ABARTICULAR MANIFESTATIONS OF ACUTE RHEUMATISM.—Most conspicuous among the abarticular manifestations of acute rheumatism are the affections of the circulatory apparatus. Principally important among these is rheumatism involving the heart. Such concurrence of cardiac rheumatism with severe forms of articular rheumatism is a frequent event, but in the milder forms of articular rheumatism cardiac lesions are less commonly witnessed. Yet this rule is not without exceptions. Severe cases of cardiac disease sometimes accompany very mild forms of articular disorder. It is impossible to determine statistically the relative frequency of the cardiac and the articular forms of rheumatism, since in a certain number of cases the existence of endocarditis remains undiscovered during life. Among children a considerable predisposition to cardiac disease is manifested, even though the external phenomena of rheumatism be of a comparatively trifling character. Of the different forms of cardiac disease, endocarditis is probably the most frequent, though this statement has been disputed.

Cardiac rheumatism is sometimes developed before the occurrence of articular lesions. It may be sometimes recognized during the period of apparent invasion, several days before swelling of the joints appears. Sometimes cardiac disease commences and persists for months or even years before any articular disorder is manifested : it then indicates the existence of that arthritic diathesis of which it is perhaps the most conspicuous symptom. It is probable that in the acute articular form of rheumatism the cardiac localization of the disease commences with the commencement of the fever, though it is true that the audible signs of endocarditis are not generally sufficient for recognition before the end of the first week. Careful auscultation will determine the existence of alterations in the rhythm of the heart, characterized by an equalization of the sounds and the intervals by which they are separated. The cardiac pulsations may be exaggerated in force or reduced in vigor, and the number of pulsations exhibits considerable variation. The præcordial region is sometimes painful on pressure before the existence of endocardial or pericardial sounds can be recognized. While it is necessary to avoid the mistake of considering anæmic murmurs as evidence of endocardial inflammation, it must not be forgotten that there is greater danger of overlooking the existence of actual endocarditis. Auscultation of the heart should be practised every day in order to avoid failure in the discovery of cardiac disease.

Pericarditis is seldom manifested before the commencement of articular inflammation: its existence usually becomes evident during the second week of acute rheumatism; sometimes its commencement dates from a later period of the disease. It is often difficult to decide whether an endocardial murmur or a pericardial friction-sound is the result of recent inflammation or of an old lesion. When the auscultatory sounds are fully developed at the commencement of an attack and remain unchanged throughout its course, they are probably occasioned by antecedent disease. A recent inflammation may be inferred when the observer can trace the origin, course, and change of the auscultatory signs.

Cardiac rheumatism, like the articular form of the disease, is subject to great variation in its course, duration, severity, exacerbations, and termination. The consequences of cardiac disease are, however, more likely to be permanent than the lesions which involve the articulations. Sometimes, however, an endocardial inflammation may subside with the subsidence of the articular disorder, and leave behind no trace of its existence. Sometimes, on the contrary, the endocardial changes persist with increasing severity, and result in the establishment of a chronic cachexia, together with all the consequences of chronic valvular disease of the heart. While the majority of mild cases terminate in complete recovery, it is probable that about 40 per cent. of the cases of cardiac rheumatism result in permanent disease of the circulatory apparatus. Acute articular rheumatism therefore constitutes one of the most frequent causes for the incidence and development of cardiac disease.

Much discussion has arisen regarding the determining cause of cardiac inflammation in connection with acute rheumatism. Cardiac complications occur more frequently in childhood than during adult life. For those who accept the infective origin of rheumatism it is easy to explain the occurrence of endocarditis by the presence of infective agents or an infective virus in the blood; but until such contagion has been demonstrated this explanation must remain a matter of hypothesis. The presence of certain micro-organisms in the inflamed tissues is what may be usually observed whenever inflammation exists in any part of the body, but their special incidence upon the valves of the heart remains unexplained.

The arteries do not directly participate in the changes due to acute articular rheumatism, but they are liable to a chronic form of endarteritis and periarteritis, and to atheromatous degenerations which are probably connected more or less indirectly with the pre-existence of rheumatic disease. The veins are more frequently invaded by acute rheumatic inflammation than the arteries, but such forms of phlebitis are usually associated with chronic and anomalous manifestations of rheumatism rather than with its acute articular variety.

Cerebral Rheumatism.—It is chiefly in the subarachnoid network in the vessels of the pia mater that the lesions of cerebro-spinal rheumatism usually exist. The dura mater is rarely involved. When pachymeningitis exists, it is probably due to chronic alcoholism or to other diseases which antedate the attack of rheumatism. In certain cases after death from cerebral rheumatism the only visible lesion is an excessive anæmia of the cerebral substance and of its membranes. Various convulsive or paralytic conditions, indicative both of irritation and of paralysis, may be produced by an anæmic condition of the brain.

The most conspicuous lesions of the cerebro-spinal structures are, however, of a congestive nature. The subarachnoid spaces and the serous cavities within and without the brain are distended with serum which is sometimes tinged with blood. The veins of the cerebral envelopes and of the choroid plexuses are engorged with blood. If the disease has been somewhat prolonged, local hæmorrhages are visible upon the surface of the cerebrum, especially upon the lateral and inferior portions of the hemispheres. The cerebral substance itself appears congested, the gray matter assumes a rose color, and incision of the centrum ovale is followed by the appearance of numerous points of blood upon its white surface. In many cases microscopical examination indicates the existence of actual inflammatory exudation upon various points of the meningeal surfaces or upon the pia mater and gray substance of the cerebral hemispheres. Occasionally actual suppuration may occur in the cavity of the arachnoid, but in such cases the course of the disease is generally protracted and obscure. Usually the effusions are of a serous character, and are dependent upon an obstructed condition of the circulation. In the majority of cases it is probable that, like the corresponding lesions in the articulations, the pathological changes in the brain and in its membranes are of a nature that permits of their rapid development and subsidence, so that in case of recovery they may often leave no trace of their occurrence.

It was formerly supposed that the incidence of cerebral rheumatism could be explained by the assumption of a metastasis of the local manifestations of the disease from the joints to the intracranial tissues; but it is now admitted that cerebral rheumatism is not to be regarded as a metastasis, but as a particular exhibition of a general disease.

The extraordinarily high temperature that frequently accompanies cerebral rheumatism has been supposed to be the cause of the manifestations of cerebral disorder; but while it is true that an excessive temperature is potent to produce disorder of structure and function in the cerebral tissues and other organs of the body, the cause of such high temperature in rheumatism must be explained by something different from mere elevation of bodily temperature. It is also a fact of observation that in many cases of cerebral rheumatism no such inordinate rise of temperature is observed. Only when the regulative centres are crippled is the phenomenon exhibited.

In certain cases of cardiac rheumatism delirium and other cerebral disorders are sometimes witnessed; but such cardiac disease cannot be designated as the sole cause of delirium and mental perversion, since disturbances of cerebral function are sometimes experienced without the coexistence of cardiac disease in any appreciable form. In certain cases, however, there is an undoubted connection between cerebral rheumatism and cardiac disease that can be explained by the occurrence of cerebral embolism or other accident dependent upon a morbid condition of the circulatory apparatus.

Many authors have expressed the opinion that cerebral rheumatism is due to a species of oscillation between the brain and the articulations by which the disappearance of articular inflammation was followed by local disturbance of a cerebral character. This opinion is based upon the fact that in many cases of cerebral rheumatism pain and swelling disappear from the joints. To this explanation, however, may be objected the fact that such disappearance of articular symptoms is not always complete, and also the fact that the course of cerebral rheumatism is sometimes interrupted by remissions and exacerbations that are not coexistent with any return of external manifestations of the disease.

It hardly needs to be stated that the reference of cerebral rheumatism to the use of certain therapeutical agents, like quinine, is no longer tenable, and serves only to be ranked with those exhibitions of popular ignorance which refer all disagreeable morbid symptoms to the particular form of medication which may chance to coincide with the special manifestation of the disease that is the subject of complaint.

Cerebral rheumatism is not a common disorder. It scarcely exceeds 3 or 4 per cent. of the cases of acute articular rheumatism. Like the cardiac form of rheumatism, the occurrence of cerebral disorder is subject to considerable variation in the frequency of its occurrence. Certain years and certain seasons are especially fruitful in this form of the disease: it is more frequent among men than among women, but this is probably due to the greater degree of exposure to inclement weather and to alcoholism on the part of the male sex. It is between the ages of twenty and forty years that this variety of rheumatism is most commonly witnessed. It is sometimes experienced by children, but they are most liable to other forms of nervous disorder.

The influence of the season of the year is very obscure, and has never been reduced to a statistical form. It is, however, a fact that exposure to cold may act as an exciting cause of the morbid manifestations, just as it may apparently determine other localizations of rheumatism.

Chief among the causes of cerebral rheumatism must be ranked that vague and unexplained condition which is designated by the term "cerebral predisposition." It is among patients who have either inherited or acquired a predisposition to cerebral disorder that this particular variety of rheumatism is manifested. It is among irritable, excitable, melancholic, epileptic, and hysterical subjects that cerebral manifestations are most to be feared during the course of acute articular rheumatism. The occurrence of alcoholism, lead-poisoning, uraemia, and the previous existence of chronic cerebral lesions of any kind, are also to be considered as predisposing causes of cerebral rheumatism. It is necessary, however, to avoid the error of mistaking for cerebral rheumatism the concurrence of an ordinary paroxysm of hysteria, or an attack of delirium tremens with acute articular rheumatism.

The manifestation of cerebral symptoms is usually witnessed during the height of the articular disease, ordinarily between the fifth and twentieth days of its course. They may be developed in connection with any variety of the disease, though more frequently witnessed in its severer forms. The onset of the attack is often very sudden, but sometimes it is foreshadowed by the occurrence of unusually high temperature, miliary eruption, profuse perspiration, and slight nocturnal delirium, together with headache and inordinate sensitiveness about the affected articulations. In certain cases there are sudden disturbance of vision, alteration of the pupils, vertigo, hallucinations, difficulty of articulation, subsultus tendinum, and sudden disappearance of articular pain and sensibility.

The **symptoms** of cerebral rheumatism differ very little from the ordinary symptoms of cerebral disorder from other causes. Headache is a common symptom. Vomiting is not often witnessed. Constipation frequently occurs both in the cerebral and in the articular forms of rheumatism. Delirium is variable in its form and intensity: it often appears at night and disappears during the daytime; it can assume a mild form, especially among children, but it is frequently violent and furious, and accompanied by a disposition to noisy utterance and struggling efforts to get out of bed and to escape from the attendants of the sick-room: when accompanied by considerable increase of temperature the symptom is more serious than if the temperature remains moderate or exhibits some degree of depression. The delirium tends to become continuous and excessive, sometimes accompanied by convulsions, and merging finally into a condition of coma, followed by death. In milder forms of the disease the delirium assumes a less violent character, and consists rather in perversions and enfeeblement of the intellectual faculties than in their exaltation and speedy abolition. This form sometimes resembles delirium tremens or melancholia accompanied by delusions of persecution and other mental perversions of a depressive character. In certain forms of cerebral rheumatism delirium constitutes almost the only manifestation of cerebral disorder.

The condition of coma is usually developed after the occurrence of delirium or convulsions, but in certain cases it appears suddenly, without previous phenomena, is accompanied by an inordinately high temperature, and rapidly advances to a fatal termination.

Convulsions are usually associated with other characteristic symptoms of cerebral disturbance, but sometimes they constitute the only conspicuous phenomenon, and may speedily lead to a fatal termination. The circulation is frequently disturbed during the course of cerebral rheumatism, and the pulse is liable to considerable acceleration, independently of disturbances that may be connected with concurrent cardiac disease.

The extraordinary elevation of temperature that accompanies the development of cerebral symptoms constitutes one of the most conspicuous phenomena of this variety of rheumatism.

In cases that are not speedily fatal the patient exhibits rapid emaciation and a high degree of anæmia, dependent upon disturbances of nutrition that are consequent upon cerebral disorder.

The clinical varieties that are exhibited by cerebral rheumatism are as numerous as the patients themselves, but they may be arranged into three principal groups, of which the first includes those rapid and violent manifestations of the disease that are principally characterized by coma, convulsions, and death or by violent delirium rapidly terminating in death. The second group includes the largest number of cases, and comprises all acute cases accompanied by cerebral symptoms, with or without excessive elevation of temperature, in the course of acute articular rheumatism. The third group includes all cases of subacute and chronic cerebral disturbance more or less directly connected with articular rheumatism. In this group are found the forms of insanity of rheumatic origin.

It is an interesting fact that similar cerebral disturbances may be associated with gout and with the mild, chronic, irregular, and secondary forms of rheumatism.

The **diagnosis** of cerebral rheumatism is seldom difficult, except in the prodromic stage or when the articular or cardiac manifestations of the disease are absent. There is little in the symptoms themselves to indicate the nature of the cause of disturbance. In a large proportion of the cases concurrent manifestations of disease elsewhere and the history of the case are needful in order to establish the diagnosis.

Cerebral rheumatism is an exceedingly dangerous disease: a large number of the cases terminate fatally. The course of the disease is often rapid, tumultuous, and speedily fatal, yet recovery sometimes occurs in an unexpected manner.

A continuous course of the disease is an unfavorable indication, while the occurrence of remissions is a favorable sign. The absence of a high temperature may be also regarded as a hopeful symptom. Of the three classes of patients above indicated, the first yields the greatest mortality, while the third affords the largest number of recoveries.

The mere presence of delirium in connection with acute rheumatism is not particularly dangerous. The occurrence of delirium or of convulsions followed by coma is of almost inevitably fatal significance.

A high temperature associated with the symptoms of cerebral rheumatism is of the gravest import. A temperature of 106° F. indicates great danger, and its further ascension is almost always fatal. During the period of convalescence and in the coma that precedes death temperatures of 110° or 111° F. are sometimes witnessed.

SPINAL RHEUMATISM.—Rheumatic inflammation of the spinal meninges may accompany the course of acute rheumatism. The character of the anatomical changes is identical with the similarly related changes in the brain and its envelopes. When a purulent exudation is present, it probably belongs to meningeal inflammation of a pyæmic character or to those nervous diseases that are accompanied by arthropathic disorders of spinal origin.

When associated with the phenomena of cerebral rheumatism the concurrence of spinal symptoms is frequently overlooked; and it is often difficult to distinguish between rheumatic pains in the extremities that are dependent upon local causes and those which are excited by central disease of the spinal cord and meninges.

It will be found convenient to group the different varieties of spinal rheumatism into three classes, of which the first includes the milder forms of the disease, characterized by spinal pain that is increased by pressure upon the spinous processes, and by indefinite pains in the lower limbs and along the larger nerves. Occasionally there is a moderate reduction of muscular power in the lower limbs, and some difficulty is associated with the evacuation of the bladder. These cases are attended by a moderate degree of fever; their duration is brief and recovery is the rule.

In the second class of cases there are high fever with an elevated temperature, severe pain in the back that is increased by movement, and symptoms that indicate active irritation of the meninges and roots of the spinal nerves. Sometimes the bladder and the rectum are paralyzed: there are various disturbances of sensation, accompanied by muscular contracture, tremor, and paresis of the lower extremities. The prognosis is less favorable than in the previous class of cases, but complete recovery frequently occurs.

The third class includes the most formidable and dangerous cases. They are characterized by intense fever, great elevation of temperature, symptoms of compression of the cord, muscular contracture, tetanic paroxysms, paraplegia, paralysis of the bladder and rectum, and are usually accompanied by corresponding cerebral complications.

Rheumatic Disorders of the Respiratory Organs.—The symptoms of coryza not unfrequently appear at the commencement of an attack of acute articular rheumatism. Similar invasion of the laryngeal structures is sometimes experienced, involving the mucous membrane, the muscles, the nerves, and the articulations of the laryngeal cartilages. Laryngeal rheumatism is accompanied by laryngeal pain, hoarseness, aphonia, and tenderness on pressure over the external surface of the larynx.

The trachea and bronchi sometimes exhibit evidence of inflammation, associated with acute articular rheumatism.

Pulmonary congestion is not an uncommon event during the course of acute rheumatism. It may be generalized and acute, or it may be partial and moderate in degree. The severe forms of the disease are characterized by sudden dyspnœa, with a sensation of great compression and painful constriction of the thorax. There are distressing cough and a viscid, bloodstained expectoration. Sometimes œdema is rapidly developed, and the patient is suffocated by a copious exudation that fills the air-cells and bronchi, escaping from the trachea in voluminous masses of foam. Death rapidly follows the development of the suffocative paroxysm.

In the great majority of cases, however, the course of pulmonary congestion is less rapid and severe. Like other forms of rheumatism, it is subject to remissions and exacerbations, and may often be relieved by therapeutic measures.

Associated with cardiac rheumatism, œdema of the lungs and various other pulmonary and bronchial complications of a passive and chronic character are not uncommon.

Genuine lobar pneumonia is not often witnessed in connection with acute rheumatism, though it is sometimes thus associated. The pulmonary manifestations which present the ordinary symptoms and physical signs of pneumonia during the course of acute rheumatism, and are rapidly dissipated, leaving no trace behind, are not to be considered as genuine examples of pneumonia, but are of a congestive and transient character, like the corresponding exhibitions of rapidly-subsiding articular inflammation. In the majority of cases pulmonary inflammation exhibits the form of broncho-pneumonia, pleuro-pneumonia, or lobular pneumonia, rather than the genuine lobar form of the disease.

Pleurisy is frequently associated with cardiac rheumatism and with the other lesions of the lungs and respiratory passages which complicate the course of articular rheumatism. Its existence is sometimes overlooked in consequence of painful manifestations involving the thoracic walls, the heart, or the lungs. The symptoms and the course of rheumatic pleurisy differ in no essential particular from ordinary forms of the disease.

THE MANIFESTATIONS OF ACUTE RHEUMATISM IN THE ALIMENTARY CANAL are principally observed in the pharynx and in the intestine: it is not uncommon for an attack of acute rheumatism to be introduced by fever, severe pain in the pharynx, and difficulty of deglutition. This is sometimes accompanied by painful swelling and difficulty of motion in the cervical muscles. A papular erythema is sometimes also visible for a short time upon the surface of the skin. These manifestations may be accompanied, or more generally followed, by articular pain and swelling. Sometimes inflammation of the tonsil proceeds to suppuration and the formation of abscess. The painful character of these pharyngeal inflammations is unusually severe, and is but slightly relieved by antirheumatic treatment. Opiates alone are capable of affording some measure of comfort during the attack.

In certain rare instances rheumatic pain is experienced in the œsophagus during the course of acute rheumatism. Gastric pain and intestinal pain are more commonly felt, but they are often overlooked in consequence of the predominance of other neighboring pains. Sometimes diarrhœa occurs, attended with pain of unusual severity. Ordinarily the liver remains inactive, and constipation is the rule during an attack of acute rheumatism.

Rheumatic peritonitis is a rare event, but it is sometimes experienced in alternation with the articular manifestations. It is characterized by intense suffering, and is not infrequently fatal.

Renal inflammation is not a common event during the course of rheumatism, unless occasioned by embolic obstruction of the renal vessels as a consequence of valvular disease of the heart. The usual form of inflammation when the kidneys are affected is a slight catarrh of the uriniferous tubules, which is of brief duration and is accompanied by a mild and transient form of albuminuria.

Inflammation of the bladder is sometimes witnessed, but it is not a frequent event. The same thing is true of urethritis and orchitis.

Various cutaneous eruptions are often observed in connection with the evolution and course of acute rheumatism. Miliaria and sudamina frequently appear as a consequence of excessive sweating, just as they accompany similar conditions in other diseases. In the majority of cases the eruptions in rheumatism exhibit the characteristics of the different varieties of erythema. They may occupy any portion of the body, and may appear at any time during the course of the disease. They are frequently tender upon pressure, and are accompanied by considerable itching. They usually disappear rapidly, and are followed by desquamation, but sometimes they develop a vesicular stage before final subsidence. The papular eruptions are generally developed upon the back of the hands, upon the fingers, lips, and nose. Erythema marginatum particularly affects the body, face, and neck. Herpetic eruptions appear upon the face. Purpuric patches are usually found in the neighborhood of the armpits and upon the lower limbs, while erythema nodosum is generally observed upon the lower limbs and over the tibia, where it simulates periostitis. The papular forms of erythema sometimes appear before articular symptoms are developed, closely resembling the eruption of roseola or scarlatina. Urticaria also frequently precedes the other local manifestations of acute rheumatism.

The occurrence of these various cutaneous eruptions possesses little prognostic significance, for they are associated with mild forms of rheumatism as well as with the most dangerous varieties of the disease. It is probable, however, that purpuric and hæmorrhagic eruptions indicate a more dangerous tendency to dissolution of the blood than the ordinary mild and transient rashes.

Diagnosis of Acute Articular Rheumatism.—The symptoms of acute articular rheumatism are easily recognized, and if careful observation of the heart and other internal organs be practised, the visceral manifestations of the disease can scarcely be overlooked. The principal points in a differential diagnosis depend upon a recognition of those forms of articular inflammation that are dependent upon infective diseases, such as scarlet fever, typhoid fever, relapsing fever, puerperal fever, diphtheria, erysipelas, dysentery, pyæmia, septicæmia, syphilis, and gonorrhœa. Rheumatism is seldom witnessed during the first years of life. The articular diseases which are then experienced are usually of a suppurative character. When rickets is accompanied by fever and swelling of the articular extremities of the bones, it closely resembles rheumatism, but in such cases the swelling is not limited to the joints, but extensively involves the bones. Acute periostitis occurring near the articulations may be sometimes mistaken for rheumatism. The surest differential indication is derived from a careful observation of the seat of swelling, which in periosteal inflammation is situated upon the articular extremities of the bone, and not in the region of the synovial capsule.

The differential diagnosis of acute gout involving many of the joints will be greatly aided by the history of the patient, though it must be remembered that gouty subjects may sometimes experience acute rheumatism, and, moreover, a rheumatic attack sometimes commences suddenly in the night, involving the great toe, which is the favorite seat of gout. In doubtful cases the uric-acid test may become necessary to decide the diagnosis.

It is not always easy to determine the nature of an inflammation that involves a single joint alone. Such inflammations are usually non-rheumatic in their character, but this rule is not without exceptions.

Prognosis.—With the exception of cerebral forms of the disease, acute articular rheumatism is seldom attended with immediate danger to life. The mortality rarely exceeds 3 or 4 per cent., even including cardiac, cerebral, and other visceral forms of the disease. The remote eventualities of the disease, such as are dependent upon chronic valvular lesions of the heart, are not included in this estimate. The prognosis in every case of acute rheumatism is therefore not without a certain degree of uncertainty, since mild forms of the disease may sometimes become suddenly transformed into most violent and dangerous manifestations of its energy. In old age acute rheumatism is less frequently experienced than in middle life, but it is more liable to be succeeded by chronic neurotic disorders of a depressing character. When rheumatism appears during infancy and early life, the prognosis is unfavorable with regard to the future, since it indicates a predisposition that will render the patient liable to subsequent attacks and to various irregular forms of the disease which may seriously compromise the health of the individual. Not unfrequently chorea is experienced by such patients, apparently as a sequel or relapse of a rheumatic attack.

At every period during the course of a rheumatic attack the occurrence of any unusual symptom or exaggeration of ordinary phenomena should be regarded as an addition to the gravity of the prognosis.

Treatment.—Acute rheumatism requires perfect rest in bed, where alone it is possible to secure that moderate action of the heart which is so desirable in a disease that is disposed to concentrate its energies upon the central organs of circulation. In order to prevent sudden refrigeration of the surface of the body during profuse perspiration it is necessary to place the patient between flannel sheets, but he should not be overburdened with excessive clothing.

Copious draughts of liquid may be allowed, in order to assuage the thirst of the patient. Lemonade and other slightly acidulated drinks, milk, and aërated waters are usually agreeable. Alcohol should not be given during the acute stage, but after the development of anæmia and debility much advantage may be sometimes obtained from the moderate use of wine or well-diluted spirits.

During the inflammatory stage of the disease animal food, with the exception of milk, should be withdrawn, but after the subsidence of acute symptoms, and especially during convalescence, a generous diet is admissible: it is, however, expedient to avoid an early return to a rich meat diet; fish, oysters, and the breast of chicken should be preferred until recovery is far advanced.

At the outset of the disease a mercurial cathartic (calomel gr. x, pulv. jalap comp. gr. x, sod. bicarb. gr. v) is beneficial; but during the acute stage of polyarthritic rheumatism active cathartics should be withheld, since their operation will only increase the distress of the patient.

The practise of bleeding, which was formerly so universally employed, is now completely abandoned. Dry cups along the spine may be applied every day with considerable benefit, and when the symptoms indicate a subacute form of spinal rheumatism, the application of wet cups is often followed by great relief.

In certain obstinate cases that do not readily yield to ordinary treatment the application of blisters in the neighborhood of the inflamed joints often affords great comfort, and is followed by a subsidence of acute symptoms.

The number of drugs that have been employed by the older physicians in the treatment of acute rheumatism is almost beyond computation. Prominent among them were calomel or calomel and opium, tartar emetic, tincture of the sesquichloride of iron, lemon-juice, extract of aconite, tinctures of cimicifuga, colchicum, veratrum viride, and guaiacum, quinine, nitrate of potassium, the alkaline carbonates, bicarbonates, and citrates, chloride of ammonium, caustic ammonia, trimethylamine, and numerous other vegetable and mineral remedies that have been employed with indifferent success. The alkaline treatment was probably more useful than any other in their hands. It consisted in the daily administration of the bicarbonate of potassium, of the citrate of potassium, or the bicarbonate of sodium, in doses of thirty to sixty grains, taken every three or four hours in an effervescing draught, until the urine was rendered neutral or slightly alkaline. For the relief of pain it was customary to administer Dover's powder in ten-grain doses every four hours. Garrod introduced the practice of giving quinine with a solution of bicarbonate of potassium. He was in the habit of giving five grains of quinine with thirty grains of bicarbonate of potassium every four hours: by this method the symptoms of the disease were more readily relieved than by the alkaline treatment alone. Duckworth considers this method especially useful in rheumatic pericarditis and in great prostration of the nervous system. In the form recommended by the English physicians this method of medication is extremely disagreeable, but it may be rendered more tolerable by administering the quinine separately in a wafer, while the alkaline salt is given in the form of an effervescent draught.

In the year 1874 Dr. Maclagan of Scotland undertook the treatment of acute rheumatism with large doses of salicin, but the unquestionable value of this remedy has been almost completely overshadowed by the introduction of salicylic acid in the year 1875. This substance, discovered in 1838, was employed as an antifebrile agent by the Swiss and German physicians

Buss and Reiss. They soon noted the remarkable effects of the drug upon rheumatic fever, and, publishing their observations, the use of the new remedy speedily became general throughout the civilized world. The new method of treatment was attended by a most astonishing effect upon the course of acute rheumatism, and the therapeutical methods which had been previously employed were soon relegated to the realm of ancient history. Patients who formerly suffered indescribable anguish for days and weeks in succession, with scarcely appreciable results from the alkaline solutions with which they were drenched and the sudorific opiates with which they were drugged, now found themselves released from pain in the course of a few hours, and able to leave their beds and to dress themselves at the end of the third day. Opium and morphine were no longer needed to procure sleep, and the treatment of acute articular rheumatism was transformed into a positive pleasure for the physician, instead of being the wearisome and heart-sickening drudgery so often experienced in former times.

Pure salicylic acid produces the most prompt and certain effects in the treatment of rheumatic fever, but its low degree of solubility has largely interfered with its use: for this reason the salicylates of sodium, of potassium, and of ammonium have generally replaced the acid. Salicylic acid may be, however, administered in pills, capsules, or effervescing draughts. The salicylates may be given in a simple aqueous solution or in lemonade: whichever preparation is selected, the dose should be from ten to fifteen grains every two hours until the ears begin to hum and articular pain and swelling subside; the intervals between the doses may be then lengthened to three or four hours, but a continuous influence of the remedy must be sustained for at least a week after the disappearance of all acute symptoms, otherwise relapses will be frequently witnessed.

Salicylic acid and the salicylates are powerful agents for the reduction of temperature in all forms of fever, but in addition to their antipyretic action they exert a special anodyne influence over the painful symptoms of rheumatism. Their influence upon the tumefaction of the affected joints is usually conspicuous, and is due to their effect upon the nervous system, controlling the circulation and transudation of liquid from the capillaries about the joints.

The effect of the salicylates upon cardiac and other visceral lesions in acute rheumatism has been debated at great length. It is now generally admitted that these remedies possess great efficacy for the prevention of such lesions, but that they are comparatively powerless to cure them when once established. Many of the failures to prevent cardiac lesions have been due to an inefficient and timid administration of the drug during the early stage of the disease.

The manner in which salicylic acid produces its favorable influence is not yet fully understood. It is very efficient in the elimination of uric acid and the biliary excreta, but its action is not merely eliminative: it is specifically anodyne, and is marked by great power to regulate certain disordered functions of the nervous system, exercising a controlling influence over the liberation, distribution, and discharge of heat. It is also probable that it exercises a powerful influence over the processes of metabolism in the tissues.

The failure of the salicylates to produce amelioration of the symptoms of acute rheumatism is frequently due to an inefficient administration of the remedy, but in a small number of cases this explanation cannot be urged: it is probable that some of these cases are not real examples of rheumatism, but are of gonorrhœal or septic origin. Gouty patients and sufferers with febrile exacerbations of rheumatoid arthritis contribute a certain proportion of these failures. Sometimes, however, especially among elderly people, the disease is not amenable to the salicylates, and is more favorably influenced by the administration of quinine and the alkaline salts, as recommended by Garrod. Anæmic and adynamic patients are always benefited by a tonic and stimulant treatment that improves the circulation and sustains the heart. In such cases capsicum, guaiac, strychnine, arsenic, iron, and alcohol are of great service. It is in acute and sthenic forms of rheumatism that the salicylates are most conspicuously useful.

The prolonged and vigorous use of the salicylates is sometimes attended with disagreeable consequences. Gastric irritation may be thus produced, and certain patients with weak hearts find these drugs too depressing in their character. Occasionally headache or a peculiar delirium, accompanied by hallucinations identical with those of delirium tremens, may be experienced. It is doubtful whether albuminuria and hæmorrhagic effusions can be correctly ascribed to the use of salicylates, since those accidents are sometimes witnessed in the course of rheumatism when these preparations have not been administered. It is stated on good authority that these toxic effects are seldom if ever witnessed when perfectly pure preparations are employed. For this reason the oil of wintergreen and salicylate of sodium that has been prepared with it have been recommended by experienced practitioners. There can be no doubt that the large doses in which the impure drug is sometimes given must exercise irritant and unfavorable effects; but with caution in this respect no danger need be apprehended. Salicylate of sodium is the remedy most frequently employed, on account of its ready solubility and its comparative freedom from irritating influences upon the mucous membrane of the stomach. Salol is by many considered fully equal to the salicylate of sodium, but its insolubility and its decided effects upon the kidney, together with its comparatively high price, have combined to greatly restrict its employment.

Benzoic acid and the benzoate of sodium have been given in the same doses and same manner as salicylic acid and the salicylates, but they possess no advantages over those remedies. Other antipyretic agents—antipyrine, acetanilid, and phenacetin—possess considerable power to alleviate the painful and febrile symptoms of acute rheumatism, but they are less efficient than the salicylates, and antipyrine is far more depressing in its effect upon the heart.

The inflamed and tumefied joints should be wrapped in flannel or in cotton wool covered with oiled silk. It is also important that the affected joint should be kept as motionless as possible. It sometimes becomes necessary to apply a well-padded splint for the purpose of preventing motion of the inflamed surfaces. Since the introduction of the salicylates the use of evaporating lotions and anodyne applications has been rendered almost wholly unnecessary. The same thing is true of hypodermic injections of carbolic acid and other anodynes in the neighborhood of the painful articulations. When other treatment fails, the application of blisters near the joints is usually quite effectual. In certain obstinate cases involving the wrist frequent fomentations with water as hot as can be tolerated are very serviceable.

The occurrence of endocarditis does not require any special treatment. It was formerly customary to give mercury in such cases, but the practice has now been generally abandoned.

Pericarditis, in like manner, does not require special medication unless the heart-sounds indicate cardiac weakness, in which case salicin may be substituted for the salicylates, and capsicum or camphor may be prescribed. If the pericardial sac be greatly distended, the operation of paracentesis must be performed by passing the needle of an aspirator through the thoracic wall in the fourth or fifth interspace, near the left border of the sternum. During the later stages of pericarditis a blister may be applied to the præcordial region, and iodide of potassium may be given for the purpose of promoting absorption.

The occurrence of pneumonia requires the same treatment as that which is ordinarily employed in the treatment of pulmonary inflammation.

Pharyngeal and tonsillar inflammation is best treated by the administration of salicylic acid in an effervescent draught. The ordinary gargles and topical applications should be also employed, such as—

℞. Zinc. sulph., ʒj;
Liquor calcis,
Glycerin, *āā.* ℥j.—M.
Sig. Apply with a probang every two hours.

The occurrence of cerebral rheumatism accompanied by an inordinate elevation of temperature demands the employment of baths, according to the method so generally useful in typhoid fever. The patient must be placed in a full bath at the temperature of 90° F., and the water should be cooled to 70° F. or even as low as 60°. The patient should remain immersed in the water, and should be rubbed as thoroughly as possible, until he complains of cold or until the temperature of the body has fallen to about 100° F. He should then be placed in bed and be rapidly dried in a blanket, receiving at the same time a cup of warm beef-tea and, if necessary, an alcoholic stimulant. Reduction of temperature, cessation of delirium, and natural sleep follow this method of treatment. The baths should be repeated as often as the temperature approaches 105° F. When the conveniences for the administration of cold baths cannot be obtained, recourse must be had to cold packs and rubbing with ice-water. It will be always necessary to explain to the friends of the patient the danger of the situation, together with a positive assertion of the benefit that may be expected from refrigeration, since popular prejudices are strongly opposed to the method.

SUBACUTE ARTICULAR RHEUMATISM.

This form of the disease is characterized by articular lesions similar to those which are encountered in the acute form, but their evolution is less speedy, cardiac and other visceral complications are less frequently observed, and convalescence is more protracted than in the acute disease. The local articular manifestations are less painful, the large joints are not invaded as often as the small joints of the extremities, but the disease is much more obstinate and tedious than when it assumes the acute form. The subsidence of local disorder proceeds very gradually, and is often imperfectly accomplished, so that stiffness of the joints and adhesion of the articular surfaces are more likely to persist as a permanent result of the disease. Visceral lesions are less frequently encountered than in acute articular rheumatism, but when they do occur they are quite as complete and as formidable as when they accompany the acute disease. There is no uniform relation between the intensity of the articular manifestations and the severity of the visceral lesions: a moderate degree of external disorder may be accompanied by the most dangerous alterations of structure in the heart and other internal organs. In like manner, though febrile symptoms are usually insignificant, the blood becomes as completely impoverished as in acute articular rheumatism. The course of subacute rheumatism is quite continuous, though subject to partial remissions and moderate exacerbations: there are nothing like the rapid changes and migratory character of acute rheumatism. The duration of the disease is usually prolonged from six weeks to several months.

Subacute articular rheumatism must not be considered as a radically different disease from the acute form. The characteristic variations are founded upon individual peculiarities of constitution and temperament rather than upon differences pertaining to the cause of the disease. It must be admitted, however, that in many cases the symptoms and behavior of the disorder closely resemble those of the forms of rheumatism that are dependent upon a previous infection, such as gonorrhœal rheumatism. In the present state of ignorance regarding the ultimate cause of rheumatism in general it is impossible to speak with decision regarding these matters.

It is often difficult to distinguish subacute rheumatism from other chronic articular diseases, from secondary rheumatism, from chronic rheumatism in its stages of exacerbation, and from subacute varieties of gout that involve the small joints. The effects of colchicum and the application of the uric-acid test will often furnish the means for discrimination between rheumatic and gouty disorders, and the course and termination of the disease will distinguish it from genuine chronic rheumatism, while attentive consideration of the history will aid in the differential diagnosis between subacute articular rheumatism and other secondary arthropathies.

The **prognosis** is less serious than in acute rheumatism, but it must not be forgotten that the course of the disease is usually longer and less amenable to treatment.

Treatment.—The salicylates and other antagonists of acute rheumatism are less efficient in subacute forms of the disease. General constitutional treatment and restorative measures are of great importance. For this purpose the administration of iodide of potassium and quinine is most effectual. Colchicum is of more value when there is considerable effusion in and about the joints. The local treatment of the tumefied articulations is of the greatest importance. The affected joints should be supported by the application of proper apparatus, but passive movements must be frequently employed in order to prevent ankylosis. Hot fomentations, flying blisters often repeated, tincture of iodine, wet cups when they can be applied, and sometimes the actual cautery, are of service.

CHRONIC ARTICULAR RHEUMATISM.

CHRONIC ARTICULAR RHEUMATISM comprises a group of slowly-developing and long-continued affections of the articular or periarticular structures that are dependent upon the same causes and changes by which other forms of rheumatism are produced.

Etiology.—Chronic rheumatism is a disease that occurs rather later in life than acute articular rheumatism. It may, however, occur at any age, though the years between forty and sixty afford the greatest number of cases. The disease is observed more frequently among women than among men. Hereditary influences operate powerfully to favor its occurrence.

Among the external causes of chronic rheumatism, cold, damp, poverty, and social misery are powerful adjuvants in the development of the disease. Disorders of the sexual apparatus, especially gonorrhœa, are not unfrequently discovered among the predisposing causes of chronic rheumatism, though gonorrhœa is more frequently followed by subacute articular rheumatism than by the genuine chronic disease.

Pathological Anatomy.—In many cases of long duration the articular lesions do not exhibit any great degree of severity. The inflammatory changes are of a superficial character, yet sometimes they are sufficient to produce contraction of the aponeuroses and tendons, so that the joints become stiffened and deformed. The changes, however, are not as deep-seated nor the bones so profoundly implicated as in rheumatoid arthritis, which in some respects the disease closely resembles. The ulnar deflection of the fingers and the peculiar alternate flexion and extension of the successive phalanges have been described by Jaccoud under the title of *rheumatisme chronique fibreux.*

In many instances chronic rheumatism apparently originates in an acute attack, which becomes unusually prolonged and gradually degenerates into the chronic form, very much as chronic bronchitis or chronic endocarditis may be related to acute attacks of those diseases. In such cases the articular lesions become more or less permanent, and do not yield to the action of the salicylates.

Symptoms.—Chronic articular rheumatism frequently exists without any conspicuous visible signs in the affected joints. The patient, however, complains of pain, which is aggravated by exposure to cold and damp weather. The joints that are most exposed to excessive fatigue during the ordinary avocations of life serve as favorite seats for the localization of the disease. The large joints are invaded as well as the smaller articulations without much regard to symmetrical or peripheral distribution, such as may be usually observed in rheumatoid arthritis. The affected joints are subject to exacerbations of swelling, heat, and pain, but in many cases they are merely stiffened without visible swelling during the greater part of the time. Their movement is attended with a peculiar creaking sound, which differs from the grating noise that is audible in old cases of rheumatoid arthritis.

Chronic endocarditis sometimes is associated with chronic articular rheumatism, but such association is less frequent than with the acute form of the disease.

Treatment.—Warm clothing and the avoidance of exposure to cold, damp weather are the principal prophylactic measures against chronic rheumatism. The local application of iodine paint upon the affected joints is useful. They may be also benefited by careful friction with neats'-foot oil, accompanied by judicious movements according to the Swedish method. For internal medication iodide of potassium and quinine, or quinine with citrate of potassium, or the ammoniated tincture of guaiacum, are the most useful remedies. Ichthyol has been alternately recommended and decried as a remedy for the disease. The salicylates are useless. General constitutional treatment of a character that improves the quality of the blood and invigorates the general health will greatly assist in the restoration of the patient. In many cases a long-continued course of cod-liver oil has been found serviceable. Frequent hot baths, accompanied by general and local manipulations during the bath, are of great service. The waters of certain saline, sulphurous, and arsenical springs have been highly recommended, but the thermal springs are most efficient, because of their temperature rather than by reason of the mineral constituents of their waters. The temperature of the bath should be constant, and every precaution should be taken to avoid exposure to a chill either during or after the bath.

ABARTICULAR RHEUMATISM.

"Abarticular rheumatism" is a convenient term that has been employed for the purpose of designating that extensive group of symptoms which, while dependent upon the same causes with articular rheumatism, are displayed in those organs and tissues that are not connected with the joints themselves. In this class may be therefore included the forms of visceral rheumatism that have been already considered, but there is in addition a large number of phenomena which may occur independently of articular disease in subjects who never exhibit any symptoms of such disorder. It does not follow, however, that such patients may not at some time exhibit articular symptoms in addition to the ordinary abarticular manifestations which they usually experience.

Rheumatism of the Skin.—Allusion has been already made to the coexistence of certain cutaneous eruptions with acute articular rheumatism, but in addition to these special and accidental symptoms of the general disturbance that accompanies an attack of acute rheumatism there is an extensive class of skin diseases that are particularly related to the rheumatic predisposition. These cutaneous disorders sometimes appear in connection with other symptoms of rheumatism, but in certain cases they exhibit a species of alternation with the characteristic phenomena of the disease. According to Besnier, the cutaneous eruptions that are specially related with acute rheumatism are the various forms of erythema, urticaria, purpura, hydroa, and pityriasis. These constitute the cutaneous symptoms of acute rheumatism, though they are by no means universally allied with that disease. It is only when they occur in connection with a rheumatic predisposition or with distinctly rheumatic phenomena in other parts of the body that they are entitled to consideration as integral parts of the general rheumatic outbreak. Erysipelas is not to be considered a rheumatic disease, though it may occur as a complication of rheumatism or may be experienced as a secondary consequence of a rheumatic attack.

In addition to the acute eruptions above mentioned, there is a considerable number of chronic cutaneous diseases which, though occurring among patients who exhibit no rheumatic predisposition, are nevertheless often witnessed under such circumstances and in such association with other rheumatic phenomena that their dependence in certain individual cases upon the causes of rheumatism cannot be denied. Among these may be mentioned a dry and circumscribed form of eczema, sycosis involving the upper lip, various forms of psoriasis occupying the palms of the hands as well as the feet and the neighborhood of the genital organs, certain varieties of acne, and the species of prurigo that is popularly known as the "winter itch." These disorders are sometimes benefited by treatment that is addressed to the rheumatic diathesis, but they are not always amenable to such treatment; and in many instances some of them are curable by local applications without regard to any constitutional condition. It is only when clearly associated or alternating with rheumatic disorders that such cutaneous manifestations can claim the title of rheumatic diseases of the skin.

RHEUMATISM OF THE VASCULAR APPARATUS.—A notable connection between endarteritis and the arthritic diathesis in the widest acceptation of that term has long been remarked. It was the opinion of Cornil that calcareous degeneration of the vascular walls is dependent upon gout, while rheumatism produces fatty degeneration of the larger arteries. Guéneau de Mussy has shown that fully one-half of the cases of chronic arteritis are dependent upon arthritic causes, and that by far the larger portion of these arthritic causes are rheumatic rather than gouty.

The effects of rheumatism upon the venous system are indicated by the occurrence of varicose veins in the lower limbs and dilatation of the hæmorrhoidal veins and of the little veins upon the alæ nasi and malar portions of the face. Sometimes similar festoons and rosettes of dilated capillaries are visible in the skin upon the lower extremities.

RHEUMATISM OF THE EYE seldom occurs in connection with acute articular rheumatism, but it is sometimes witnessed in chronic rheumatism and with secondary forms of the disease. There is nothing, however, in the characteristics of the inflammatory lesions by which they can be distinguished from simple inflammations unconnected with a rheumatic cause.

MUSCULAR RHEUMATISM.—Very little is known regarding the exact seat and character of the pathological changes that accompany muscular rheumatism. The mode of its course and its intimate connection with other phenomena of rheumatism render its nature unquestionable. It is probable that the seat of the affection is located in all the tissues that unite to constitute a muscle. The most prominent characteristic of the disorder is the existence of pain, that is excited or aggravated by every form of motion that involves the affected muscle or group of muscles.

Muscular rheumatism is a very common disease: with variable degrees of severity it accompanies the majority of acute and chronic cases of articular rheumatism. It is also often experienced as an independent affection. It occurs without much relation to the season of the year, and it is probably as common in the tropics as in the temperate zone, since it prevails wherever perspiration is liable to be suddenly suppressed by exposure of the body to cold air. It is a disease of all ages, though in its persistent and chronic forms it affects old people rather than children and young adults. Mere difference of sex probably exerts no other influence upon the liability to the disease beyond the fact that men are more subject to exposure than women. Hereditary traits and the possession of the rheumatic diathesis exercise a predominating influence in the causation of the disease.

Sudden refrigeration of the body during perspiration is the principal exciting cause of muscular rheumatism. Unilateral chilling of the surface, such as is experienced when the patient sits beside an open window in a draught of cool air, is thought to be especially prejudicial.

Very conspicuous is the influence of injuries, strains, and over-exertion of the muscles. The muscles that have been thus over-excited are most liable to invasion by the disease: for this reason its manifestations are frequently localized in the lumbar region, in the shoulders, or in the muscles of the extremities, according to the previous occupation that may have determined the particular character of muscular exertion before the attack.

The pathological changes that exist in muscular rheumatism are not well understood, since the disease is rarely fatal and the opportunity for anatomical study is seldom enjoyed. In fatal cases of acute articular rheumatism the affected muscles exhibit the changes characteristic of inflammation. The muscular fibres are swelled and granular or in a state of vitreous degeneration. It is probable that slight manifestations of a similar tendency might be discovered in ordinary muscular rheumatism were the opportunity afforded for investigation. In certain cases there is an evident atrophy of the muscles—a fact that indicates an affection of the trophic nerves by which the nutrition of the muscles is controlled.

Among the clinical symptoms of muscular rheumatism, pain is the most conspicuous. It is excited by every muscular contraction, by pressure, by variations of temperature, and by modifications of the electric, barometric, and hygrometric conditions of the atmosphere. Pain is frequently worse at night, and is aggravated by the warmth of the couch. It is sometimes lancinating and paroxysmal; in other cases it is deep, dull, and fixed; while in still other cases it is movable and transient, flitting from place to place with surprising rapidity.

The affected muscles do not often exhibit any visible changes. When swelling, tenderness, heat, and redness are apparent, the muscle is probably invaded by some other form of inflammation. Sometimes tenderness is not experienced on pressure over the entire muscle, only certain fasciculi being affected, while others escape.

The affected muscles are generally instinctively relaxed, and as far as possible maintained in a position to avoid contraction. The muscles are not actually paralyzed, but they are spontaneously relieved from duty in order to avoid the pain that would accompany their movement. Genuine rheumatic paralysis, when it occurs, is the result of lesions involving the nerves that are distributed to the muscles.

Fever is only observed in certain acute cases or when associated with acute articular rheumatism. Sometimes in chronic cases the exacerbations of pain are accompanied by a transient febrile movement that is especially severe at night.

Acute muscular rheumatism is generally manifested in a single muscle or group of muscles, such as the sterno-cleido-mastoid, deltoid, or lumbar muscles. The chronic forms of muscular rheumatism are much more widely distributed, involving many muscles at the same time or in rapid succession. Wherever muscular tissue exists, there the manifestations of the disease may be experienced. In the majority of cases the striated muscles are the seat of the disease, but occasionally non-striated muscular fibres, like the muscles of the œsophagus and of the bladder, give signs of participation.

The **duration** of muscular rheumatism is exceedingly variable. Brief and acute cases rapidly recover, but chronic forms of the disease may continue interminably.

The **diagnosis** of muscular rheumatism is determined by observation of pain which is aggravated by muscular contraction rather than by pressure upon the muscular substance. In muscular rheumatism there is absence of the painful points that are characteristic of neuralgia, and there is no real paralysis and contracture, such as are observed in cases of neuritis. In certain cases the suddenness of the attack and the rapid development of stiffness and immobility of the affected parts point to rheumatic rather than to any other cause for the painful manifestations, as when a stiff neck or a crick in the back or a painful stitch in the side are suddenly developed.

So far as danger to life is concerned, the **prognosis** is good, but the occurrence of muscular rheumatism, especially in young children, is a serious indication of the existence of the arthritic diathesis, rendering the prognosis unfavorable so far as future manifestations of that predisposition are concerned. When repeated attacks are experienced there is danger of the development of a cachectic condition or of local muscular paralysis or contracture as a consequence of participation of the nerves in the disease.

Treatment.—Severe forms of muscular rheumatism require the administration of anodynes and opiates. A hypodermic injection of morphine frequently serves to arrest, as if by magic, the painful manifestations of the disease. So grateful is this experience that the danger of thus contracting the opium habit must not be overlooked. Sometimes hypodermic injections of pure water afford equal relief, but this method is attended with considerable uncertainty. Warm applications generally afford considerable comfort. Rubber bags filled with hot water, bags of hot sand, or of heated salt—in fact, anything that can retain heat for a considerable time—will afford relief. The old-fashioned expedient of covering the affected muscles with several layers of flannel, and then passing a heated flat-iron backward and forward over the painful spot, is an excellent method for relieving the dull pain that is sometimes so distressing. The use of electrical brushes, massage, cupping-glasses, and friction with stimulating liniments is frequently beneficial, but these measures are all sometimes followed by failure. Mustard plasters and flying blisters are very effectual for the relief of pain. In many instances the promotion of perspiration is exceedingly beneficial: for this purpose the Turkish bath or an alcohol sweat is of great value. In certain cases, however, these expedients are unsuccessful, and it becomes necessary to employ the methods that are useful in the treatment of rheumatism of the nerve-trunks; in such cases benefit can be sometimes obtained from electric baths or from weak continuous currents of electricity that are applied for many hours consecutively.

RHEUMATISM OF THE NERVES.—Multiple peripheral neuritis sometimes follows acute articular rheumatism. It is accompanied by the usual symptoms of neuritis, and is often followed by trophic changes. The ulnar nerve is a favorite seat of such rheumatic inflammation. The muscles of the hand undergo atrophy and contracture, and the skin over the phalanges assumes the dry and glossy appearance so commonly observed in other forms of neuritis.

Neuralgic pains of a rheumatic character are not uncommon, though it may be sometimes difficult to distinguish between neuralgia from simple exposure to cold and genuine rheumatic neuralgia. The history of the case and the associated symptoms must be considered in order to arrive at a probable diagnosis.

Although rheumatic neuralgia may be experienced in any nerve, it is the sciatic nerve that is most frequently thus affected. Chronic sciatica is always highly suggestive of the arthritic diathesis, and is more frequently of rheumatic than of gouty origin.

ABARTICULAR RHEUMATISM OF THE INTERNAL ORGANS.—Various chronic and subacute yet distressing affections of the brain and spinal cord sometimes have their origin in rheumatism. Chronic cerebral rheumatism is characterized by vertigo, noises in the ears, a certain amount of deafness, loss of sleep, hypochondria, failure of memory, loss of appetite, and general debility. In certain cases the symptoms of insanity of a depressive type are unmistakable. The duration of the disease may continue for months or for years: it is sometimes relieved or disappears altogether upon the occurrence of the articular manifestations of rheumatism.

Hemicrania is another manifestation of abarticular rheumatism, with which it is related in a manner analogous to its relation with gout and other varieties of arthritism.

Chorea is in numerous cases undoubtedly connected with a rheumatic predisposition: it is by many authors regarded as an abarticular manifestation that is peculiar to early life, since children suffer more frequently than adults from abarticular rheumatism.

Various phenomena of spinal irritation can be often referred to a rheumatic cause. They are characterized by pain in the back and loins at night, accompanied by nocturnal priapism that is not relieved by copulation, and by indefinite sensations of discomfort in the ovaries or in the scrotum. Sometimes these nocturnal pains are experienced in the coccyx, rectum, or bladder, and are associated with various forms of myalgia, rendering life miserable notwithstanding the general appearance of health by which the patients are characterized. A rheumatic cause for all these affections should not be hastily assumed without sufficient proof drawn from other unquestionable manifestations of a rheumatic character.

In the alimentary canal rheumatism occasionally produces painful sensations in the tongue. Paroxysmal difficulty of swallowing is sometimes experienced as a result of a rheumatic condition of the œsophageal muscles. Dilatation of the stomach, acid dyspepsia, pyrosis, flatulence, vertigo, and gastralgia are not uncommon among rheumatic patients. These symptoms frequently alternate with external manifestations of a rheumatic character or with other visceral symptoms of the disease.

Intestinal flatulence, constipation, or diarrhœa, alone or alternately and consecutively, are common symptoms of internal rheumatism. Rheumatic diarrhœa is frequently very obstinate and of long duration. It should always be suspected in cases of chronic diarrhœa for which no other cause can be discovered. Certain severe forms of intestinal colic are similarly connected with rheumatism, and are successfully treated in accordance with the indications that are thus afforded. It is probable that certain cases of hepatalgia with or without icterus, and of catarrhal jaundice, are dependent upon a rheumatic cause.

Rheumatic affections of the kidney are very ephemeral and of trifling importance: while gout inflicts serious injury upon the kidney, rheumatism is seldom followed by any important disturbances of that organ. The same thing is true of the other urinary and sexual organs. Rheumatic neuralgia of the ilio-scrotal nerves extending to, and also involving, the testicle is not uncommon.

Rheumatism of the uterus is probably a rare event, but the cervical portion of the organ is undoubtedly subject to many of the lesions and pains that are characteristic of rheumatism involving the dermal and subdermal tissues of the body. Various mucous inflammations and herpetic eruptions about the neck and cervical canal of the uterus are of this character.

Of the same character are many of the so-called herpetic manifestations within the pharynx. Catarrhal affections of the respiratory organs are usually frequent and obstinate among rheumatic subjects. Sudden and violent attacks of coryza and of hay fever are often thus related. Laryngeal and bronchial inflammations also acquire similar characteristics when they occur in rheumatic subjects. Sudden attacks of hoarseness and of spasmodic cough may be thus explained. In like manner the phenomena of asthma may be excited by causes that are dependent either upon a rheumatic or a gouty predisposition, and in many instances its paroxysms alternate with the external manifestations of cutaneous rheumatism.

Besides all the above-enumerated forms by which abarticular rheumatism reveals itself, there still remains an innumerable army of cases in which the disease lies masked under various indefinite nervous symptoms. Of these, none are persistent or pathognomonic. They are multiple, variable, and fugitive, yet persistent so far as the individual is concerned. They may be considered as nervous manifestations of an otherwise latent arthritic diathesis, upon which their ultimate dependence is often demonstrated by some unmistakable explosion of the ordinary forms of rheumatism.

Chief among the causes of these nervous manifestations of masked rheumatism must be ranked the neurotic constitution, that peculiar nervous state which is either hereditary, congenital, or acquired. A debilitated condition of particular organs of the body, and that general physical deterioration which results from debauchery, over-exertion, neglect of exercise, care and anxiety, associated with unwholesome hygienic surroundings, are common causes that favor the manifestations of masked rheumatism. The existence of such a fundamental predisposition needs only the action of the exciting causes of ordinary rheumatism to arouse a whole train of morbid symptoms which chiefly involve the internal visceral nerves. The patient becomes irritable, despondent, incapable of active intellectual occupation; his sleep is uncertain, interrupted, and unrefreshing; there is a morbid susceptibility to cold; dyspepsia, neuralgia, palpitation, vertigo, and innumerable other vague manifestations of visceral nervous disorder make their appearance. These phenomena are usually experienced during cold weather: sometimes the patient seems to be wholly relieved during the heat of summer or after a vacation in the country, but with the return of damp and autumnal weather or during the winter and chilly spring months the symptoms are renewed and persist until the return of summer. In confirmed and chronic cases, however, there is a liability to manifestation of morbid symptoms with every change of weather, so that the patient becomes a veritable living barometer, sensitive to every change of atmospheric pressure, moisture, electrical condition, and direction of wind.

The **treatment** of the condition thus indicated demands particular attention to the general health rather than to the individual symptoms of which the patient complains. Long-continued medication with small, tonic doses of quinine is often the best treatment for the debilitated constitution. With this remedy may be associated iodide of potassium in small doses, iron, and cod-liver oil. Local symptoms may be sometimes relieved by the application of dry cups and various anodyne or irritating liniments. The long-continued practice of massage is also invaluable, and hydropathic treatment should never be neglected in obstinate cases. The mineral constitution of the various thermal springs is of much less importance than the degree of temperature of the water. Warm baths, careful massage, and change of air constitute the principal advantages connected with hydrothermal treatment.

SECONDARY RHEUMATISM.—The term "secondary rheumatism" has been applied to certain forms of articular disease which occur as a consequence of particular infective diseases. Of these the most common varieties are the arthropathies that sometimes follow mumps, scarlet fever, typhoid fever, relapsing fever, puerperal fever, pyæmia, dysentery, syphilis, and gonorrhœa. Until the discovery of the actual cause of rheumatism it will be impossible to speak positively regarding the exact relation that exists between such articular diseases and genuine rheumatism. It is probable, however, that their ultimate causes are different, but that the local manifestations of the action of such various causes possess a degree of resemblance that is based upon the common identity of the tissues in which the local changes are exhibited. It is in the highest degree improbable that there can be any other point of union between scarlatinal or gonorrhœal rheumatism and genuine articular rheumatism. This view is rendered still more probable by the failure of antirheumatic remedies, like the salicylates, when administered in cases of gonorrhœal or scarlatinal arthritis. The characteristics of these pseudo-rheumatic disorders can be therefore most appropriately considered in connection with the diseases from which they respectively originate.

DISEASES OF THE BLOOD.

BY WILLIAM OSLER.

INTRODUCTION.

THE blood may be looked upon either as a fluid tissue in which the corpuscles represent the cells and the plasma the matrix, or as an internal medium bearing the same relation to the constituent tissues of the body as the external medium does to the individual as a whole. The corpuscles make up a little less than one-half of the weight of the blood, the rest being plasma. The latter contains in solution the fibrin-forming factors, various proteid substances, extractives, gases, and salts. In healthy persons the composition of the blood varies within extremely narrow limits, so well compensated are the "outgoings" by the "incomings" in the different regions of the body.

During the past half century, and more particularly during the last decade, much has been added to our knowledge of the blood and its functions, most of the advances having been gained through improved methods of histological and microscopical technique.

In the healthy adult human body there are three kinds of corpuscles to be made out: (1) the red; (2) the white; and (3) the so-called blood-plaques.

The red blood-corpuscles are homogeneous, circular, biconcave disks, averaging 7.5 μ (1 $\mu = 0.001$ millimetre) in diameter. They are non-nucleated cells, consisting of a colorless framework or stroma (discoplasma), to which is united in a peculiar way the red coloring matter, hæmoglobin (paraplasma), the combination being such as to prevent the extraction of the hæmoglobin by the serum, in which it is easily soluble, and at the same time protect it from faulty processes of oxidation.

The white blood-corpuscles, on the other hand, are larger cells, all nucleated. There are several varieties of them, differing in their size, contractility, nuclear form, protoplasmic granulation, and probably in function, although on this latter point we are as yet profoundly ignorant. Some of them are capable of active amœboid movement, which may be watched with ease in fresh blood specimens. The leucocytes will be classified and further described when we speak of the differential methods of staining them.

The blood-plaques, or blood-plates, of Bizzozero are now generally recognized as normal blood-elements. They have been described under different names, and the most various functions have been assigned to them. Thus Hayem regards them as the ordinary red corpuscles in an earlier stage, and so calls them hæmatoblasts. They are small, roundish, colorless protoplasmic disks, which in fresh blood specimens have a tendency to adhere to one another, forming little aggregations resembling bunches of grapes. They stain well in aqueous solutions of the basic anilines, particularly in gentian-violet. These clumps of blood-plates have long been known as Schultze's granule-masses. The idea of Löwit, that they are formed in part by a precipitation of globulins from the blood-plasma and in part represent portions of broken-up white blood-corpuscles, is scarcely tenable. Personal observation[1] of the blood of newly-born rats convinced me, and I think will convince any experienced histologist, that these bodies are as much independent elements as are the red or white cells. They have a diameter of from 1.5 to 3.5 μ, and, according to Schiefferdecker and Kossel, are of high specific gravity. The blood-plates must not be confounded with the so-called "invisible corpuscles" described by Norris, the latter being simply ordinary red globules deprived of their hæmoglobin. I described them as main constituents of white thrombi—an observation confirmed by Bizzozero, and still more clearly demonstrated by the investigations of Eberth and Schimmelbusch, and later by Welch.

The Origin of the Corpuscles.—Although Oppel in a recent exhaustive review of the literature of the subject has been able to refer to the contributions of over one hundred investigators in this field, it must be admitted that our knowledge of the origin of the blood-corpuscles either in embryonic or post-embryonic life is even now in an unsatisfactory and unsettled state. We shall briefly mention here the views which seem to be best founded.

During the first few weeks of embryonic life there are no blood-corpuscles formed. When they do appear they come chiefly from mesoblastic cells, of which certain solid columns are laid down—the rudiments of the future blood-vessels; the central cells become loosened and break apart, gradually accumulate hæmoglobin in their perinuclear protoplasm, and form the first blood-corpuscles. The cells lining the hollowed-out columns are differentiated to form the vessel walls. These first corpuscles, which are nucleated, are large and oval, resembling somewhat the red corpuscles of amphibians. Many workers believe in an endoblastic origin for the red blood-cells at this stage. In the latter embryonal period the question of corpuscular origin becomes more complex, and to explain it the most divergent theories have been advanced. The writers of the early half of the century were of the opinion that the liver manufactured most of the red blood-corpuscles, either from cells of its parenchyma or from the interior of its rudimentary vascular columns. The later investigators favored the idea that other organs (*e. g.* the spleen and bone-marrow) also took on a blood-building function. Bizzozero, basing his conclusions on his own work and on the investigations of Foà and Salvioli, believes that in mammals during fœtal life the circulating blood, the liver, the spleen, and finally the bone-marrow, represent successive centres for the formation and multiplication of the red cells. Howell substantially agrees with this, and thinks that the newly-forming vessels all over the body give rise to blood-cells by a softening of the central cells of the columns—a fact which has been proven at least for the vessels of the posterior limb.

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[1] Cf. *Proceedings Royal Society*, 1874; *Med. News*, 1886, Apl. 3; *Centralblatt f. med. Wissenschaften*, 1882, No. 301.

The nucleated corpuscles formed in the later embryonic period are smaller, and non-nucleated forms soon appear, so that by the fifth month in the human fœtus the majority of red cells are non-nucleated. At birth there are very few nucleated red blood-corpuscles present in the blood, and they soon disappear entirely. In the adult the red corpuscles are formed almost entirely from the nucleated red cells which are found in the red marrow of bones, discovered independently by Neumann and Bizzozero in 1868, and since then carefully studied by many prominent histologists. The transformation of these nucleated cells into the ordinary red globules probably takes place by a process of extrusion of the nucleus, although many still believe that the nucleus gradually vanishes within the cell. The number of nucleated globules in the marrow becomes enormously increased where there have been great losses of blood, and in all severe anæmias they may be found in the circulating blood. The nucleated cells may divide at least once in the bone-marrow by a karyokinetic process. It is from certain light-bordered, homogeneous, colorless marrow-cells (erythroblasts) that these true hæmatoblasts arise, which, as I[1] have pointed out, are not to be confused with leucocytes. The leucocytes probably never change into red globules, although that idea first advanced by Wharton Jones is still maintained by some authors. The view of Hayem, that the red globules develop from the blood-plaques, has not been confirmed by other observers.

As to the origin of the white blood-corpuscles still less is known. A certain number come from the lymph-glands, while others apparently have their birthplace in the spleen or bone-marrow.

In a most interesting series of articles on blood-formation Löwit regards the lymph-glands, spleen, and bone-marrow as blood-forming organs, and claims that in each, from a common mother-cell, two kinds of cells free from hæmoglobin are formed, erythroblasts and leukoblasts, the latter having amœboid movement. The nucleus of the leukoblasts is relatively large, and contains one or more lumps of chromatin connected by radiating lines with the chromatin nuclear membrane. The erythroblasts are never amœboid, and have no true nucleoli; they divide through mitosis, the leukoblasts dividing through amitosis (*divisio indirecta per granula*). His erythroblasts go over into the blood, as a rule, free from hæmoglobin. They gradually accumulate coloring matter there, and so become nucleated red blood-corpuscles; the nucleus in the main becomes disintegrated and gradually disappears by absorption.

An exhaustive review of the different theories of blood-formation, together with the results of his own experimental work, will be found in Howell's article in the *Journal of Morphology* for June, 1890. Howell maintains that the red blood-corpuscles in extra-uterine life are derived from the nucleated red cells (normoblasts) by a process of nuclear extrusion (the process in health goes on in the marrow), while in anæmia some normoblasts are allowed to pass over into the circulating blood. The nucleated red cells divide by karyokinesis, and have their origin in still less mature forms, which in their turn are derived from colorless erythroblasts, the latter having resulted from successive mitotic divisions of the well-known marrow-cells with vesicular nuclei. He believes that under certain pathological conditions with extreme anæmia the spleen may again assume its red-cell building function, which under normal conditions it gives up at birth. The view advanced by Howell, that all the white elements of the blood are developed from the small lymphocytes, cannot be regarded as proven, but most histologists agree with him in thinking that a polymorphous nucleus in the ordinary leucocyte indicates a commencing retrogressive change.

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[1] *Centralblatt f. med. Wissenschaften,* 1878.

The fact that the nucleus of the normoblasts is extruded rather than absorbed has been supported by numerous observations of the blood of anæmic patients in my wards, but as to the ultimate fate of the extruded nucleus nothing definite can at present be said.

Briefly summing up our knowledge of the hæmopoietic organs, we may say that in the adult the bone-marrow undoubtedly takes the most important part in the formation of the red globules. There is also much evidence in favor of its being a developmental centre for white blood-corpuscles. The spleen has always been regarded as a blood-making organ, yet we must admit that if we look for confirmation from experimental work we find almost no support for this theory. The lymphatic glands and adenoid tissue generally are the seats of a constant production of colorless corpuscles, but there is no proof that they stand in any developmental relation to the red cells. The liver in adult life does not manufacture blood-corpuscles, but must rather be looked upon as a seat of blood-destruction. We may say, therefore, that as to the exact origin of the formed elements of the blood we are still far from having reached any unanimity of opinion. Only a few points are definitely settled, and we must wait for further investigations to clear up the subject.

Whatever may be the mode of blood-regeneration, every clinician must have been struck with the remarkable rapidity with which, after profuse bleeding, the normal proportion of red blood-corpuscles may be restored, the new corpuscles sometimes, under favorable conditions, being manufactured at the rate of thirty, forty, or even fifty, thousand per c. mm. a day.

The lifetime of a red blood-corpuscle we do not know; it probably varies considerably. The bile-coloring matters and certain of the urinary pigments would appear to have their origin in an altered hæmoglobin—a condition which, if true, would call for the daily destruction of many red globules. So far as we are able to see, however, the corpuscles when worn out are removed from the blood before they have undergone any marked changes. Though it is impossible as yet to speak with certainty with regard to the mode of disintegration, the spleen and liver are believed to be the organs in which the red cells are broken up. Occasionally in the normal spleen, liver, and bone-marrow large cells can be seen filled with red corpuscles, and it has been thought that these are blood-destroying cells. The changes which the red corpuscles undergo in the marrow, liver, and spleen have been carefully studied by Quincke and Peters, who have seen the pigment stored up within the cells of those organs in the form of an albuminate of iron, ready probably to be used over again in the development of new corpuscles.

Blood-plasma.—The study of the blood-plasma is daily assuming a greater importance, notwithstanding the tendency to attribute all serious blood-changes to corpuscular alterations. It contains water, a certain amount of serum-albumin and serum-globulin, with small quantities of inorganic salts—sodium, calcium, potassium, and magnesium.

The investigations recently carried on in connection with the question of immunity have led to a knowledge of certain previously unsuspected properties of the serum. Grohman, Nuttall, Behring, Nissen, Buchner, and others have proved experimentally that the fresh blood-serum of mammals has powerful germicidal properties. This bactericidal power of the serum of different animals varies, and, what is still more interesting, the mixing of the serum of one animal with that of another results in the destruction of the germicidal powers of both. What the substance is that possesses these peculiar powers it is difficult to say. It must be an exceedingly unstable body, since it is rendered inactive by warming at a temperature of 55° C. for half an hour, and exposure to light also robs the serum of its germ-killing influence.

Landois some time ago pointed out that the blood-serum of one animal had a destructive action on the red blood-corpuscles of an animal of a different species—a fact which explains the worse than useless practice of blood-transfusion from lower animals to man. Buchner has shown that this "globulicidal" action of the serum is quite analogous to its germicidal action. Not only are the red blood-corpuscles destroyed by the foreign serum, but the leucocytes are killed, as shown by examination on the warm stage. Buchner further refers the antitoxic action of the blood-serum of immune animals to a quite analogous chemical action. He does not believe that these substances, germicidal, globulicidal, and antitoxic, can be simple "dead" proteids in solution, but regards them as highly complex molecular combinations which in some peculiar way are dependent for their integrity on a loose combination with salts of the alkalies. Hankin terms these bodies alexins, and believes them to be derived from the leucocytes, particularly the eosinophile variety, which secrete them on suitable provocation.

The whole question is particularly interesting from a therapeutic standpoint, and at present "blood-serum therapy" (*i. e.* the injection of the serum of immune individuals into others as a protective or curative measure) is full of promise. Some astonishing results by this method in cases of tetanus are claimed by the Italian school, and in Berlin experiments are being carried on by the Klemperers and others in connection with pneumonia and the so-called anti-pneumotoxin.

Isotonia and Hyperisotonia of the Blood-serum.—The term "isotonic solution" was introduced by Hamburger in 1886. It is well known that the hæmoglobin of the red blood-corpuscles is extracted from the stroma when blood is mixed with distilled water. If one uses in place of the distilled water a solution of sodium chloride of certain strength as a diluent, the coloring matter will not be dissolved out. The salt solution which is just strong enough to prevent the solvent action has been called by Hamburger "isotonic." Several methods of determining the isotonic concentration of the serum clinically have been devised by Landois, Hamburger, Limbeck, Lakers, and others. Of course the less the concentration of the salt solution necessary to conserve the corpuscles in any given case, the greater, if we may say so, is the *isotonia* of the serum. We say the same thing when we state that the *resistance* of the corpuscles depends upon the isotonic concentration of the serum or plasma. Strictly speaking, an isotonic serum would be one in which there were just enough salts to prevent destruction of the corpuscles at the moment. The maintenance of such a condition would, however, be hazardous, as every ingestion of liquid from the alimentary canal would result in the destruction of many red blood-cells. As a matter of fact, the plasma always contains a greater percentage of salts than is necessary to prevent the extraction of the hæmoglobin from the discoplasma; hence the origin of the term "*hyperisotonia*" of the plasma or serum.

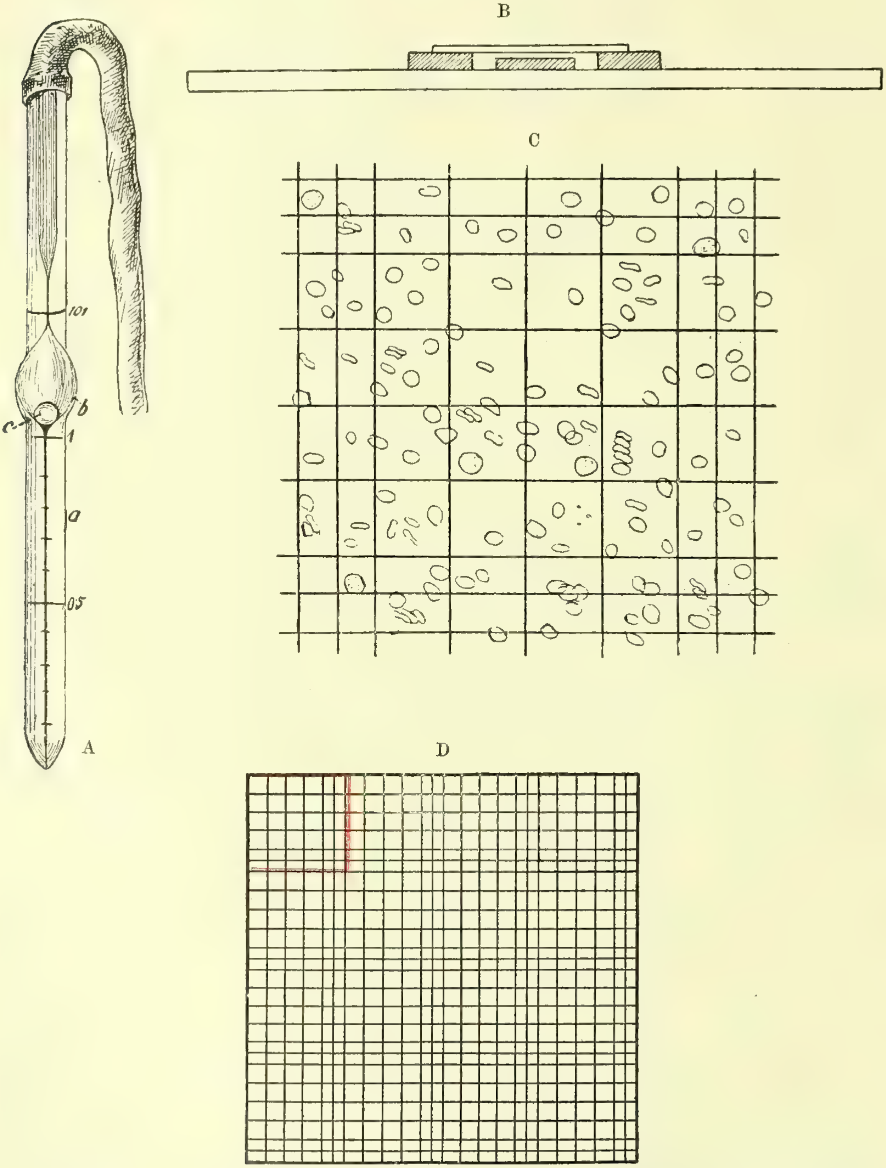
Certain other pathological alterations of the blood-plasma, such as cholæmia, uræmia, lithæmia, melithæmia, and lipacidæmia, will be considered in full in connection with the diseases in which they occur.

Before speaking of the diseases of the blood we shall discuss briefly the methods of examination to be employed, for a full appreciation of which, however, a rigid study of the text-books which contain the technical details and practical work in the laboratory under a competent instructor are indispensable.

Examination of Fresh Blood.—A microscopic slide and thin glass cover being cleaned as thoroughly as possible, and the lobule of the ear or a finger-tip (the former being less sensitive) having been washed off with alcohol or ether and thoroughly dried, a slight puncture is made with a sterile lancet or needle, and the first drop or two of blood which exudes wiped away. The cover-slip, being held with a pair of forceps, is then applied lightly to the summit of the projecting drop, care being taken not to allow the glass to come in contact with the patient's skin. Little or no pressure should be used in expelling the blood-droplet. The cover-glass is then placed at once upon the slide, and if the technique has been perfect the drop will be spread out into a thin layer. The specimen may be examined immediately with a one-twelfth inch oil-immersion lens. The form and color of the red blood-corpuscles should be noted, the relative number of whites and reds, the activity of the amœboid movement of the leucocytes, the presence or absence of parasites (the plasmodia of malaria, spirilla of relapsing fever). One can make out sometimes the predominance of certain leucocytic forms, and if there be a large number of nucleated red blood-corpuscles, they may be seen in the fresh specimen. Some care must be exercised not to allow the oil-drop to reach the edge of the cover-glass or the specimen will be destroyed.

Enumeration of the Corpuscles.—In a healthy man there should be from five to five and a half millions of red corpuscles in a cubic millimetre of blood, the number in women being normally a little under this. The white blood-corpuscles vary in number at different times of the day; thus, they are more numerous after a meal than during the digestive process. Speaking generally,

Fig. 3.



Thoma-Zeiss Blood-counting Apparatus (Limbeck).

A. Melangeur: *a*, Capillary tube in which the blood is taken; *b*, Chamber for mixing the blood with the diluting solution; *c*, Glass ball to aid in mixing the blood with the diluting solution.

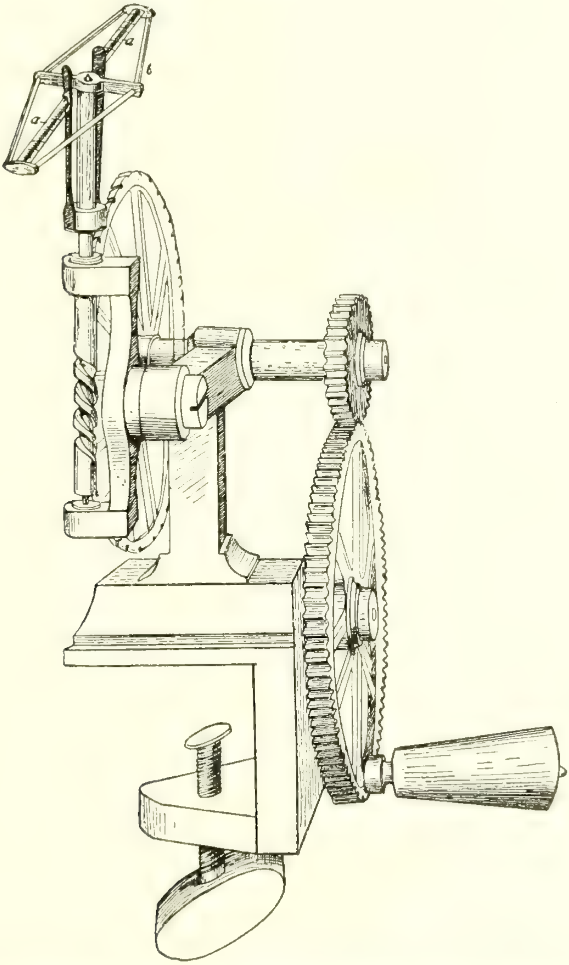
B. Cross-section of the chamber in which the blood is counted.

C. Section of the field on which the blood is counted, showing thirty-six squares.

D. Diagram of the whole field.

the average number of white corpuscles to the cubic millimetre is from 5000 to 7000. The best apparatus for counting these formed elements is the now well-known instrument of Thoma-Zeiss. (See Fig. 3.) The blood is diluted in the "mixer" (A) one hundred or two hundred times with a saline fluid of specific gravity corresponding to that of the blood. In my clinic the solution recommended by Toison is used, since it contains a little methyl violet, by which the white blood-cells are tinged and are as easy to count as the red. The drop of the mixture placed in the blood-counting cell (B) should be free from air-bubbles, and when the cover-glass has been adjusted one should be able to see Newton's rings at the margin of the drop. It is necessary to wait until the corpuscles have all settled to the bottom of the cell before beginning the count. Two whole fields (see Fig. 3, C and D, 16 large squares or 400 small squares in each) should be counted for the reds, and at least four

FIG. 4.

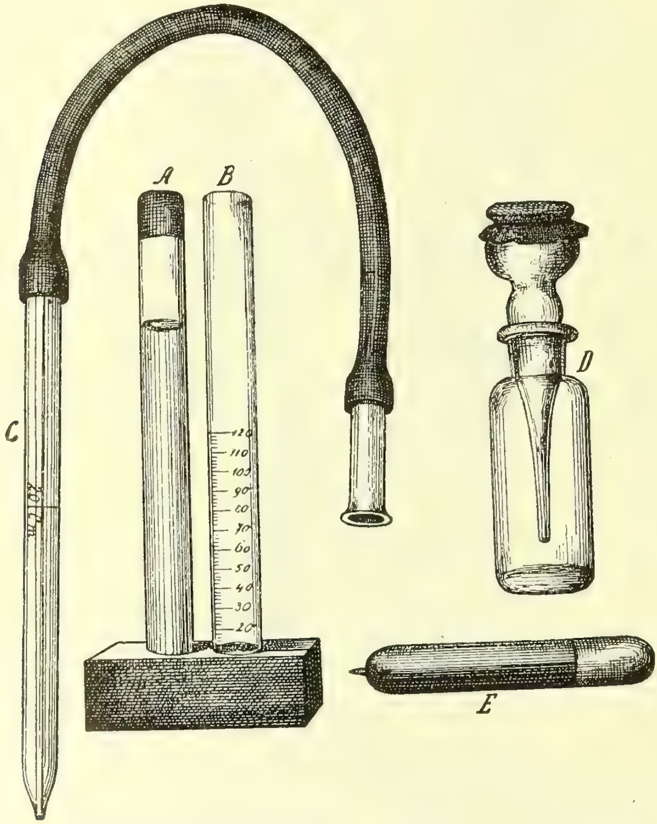


The Blitz-Hedin Hæmatokrit: *a*, Capillary tube for blood mixture; *b*, Frame for holding tubes (Limbeck).

whole fields for the whites. Although the process of counting and calculating is not a complicated one, yet careful and conscientious work is required in order that the results attained may be reliable. With the Thoma-Zeiss counter an experienced hæmatologist should not make an error of more than 2 or 3 per cent.

The centrifugal machine has been applied to blood-counting in the form of the hæmatokrit (Fig. 4) of Blitz-Hedin, and is said to give as accurate results as the hæmocytometer. Its clinical value has been definitely settled by Judson Daland,[1] working under the direction of von Jaksch in Prague. The blood is mixed with an equal volume of a 2½ per cent. solution of bichromate of potassium, or, as some prefer, of Müller's fluid, and placed in a capillary tube in the hæmatokrit. The wheel is then turned, and, the tube revolving at the rate of 9000 revolutions per minute, the red blood-corpuscles, being relatively of higher specific gravity than the white, go to the periphery, and the volume is read off on the graduated capillary tube. A long series of experiments has

Fig. 5.



Gowers' Hæmoglobinometer: *A*, Closed tube containing the standard mixture; *B*, Tube in which the blood and water are mixed; *C*, Pipette for collecting the blood; *D*, Bottle and pipette for distilled water; *E*, Lancet for obtaining the blood (Limbeck).

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[1] *Fortschritte der Medicin*, 1891, Nos. 20 and 21.

been made which show that in healthy young men the red blood-corpuscles occupy 44 to 66 parts in 100 volumes of blood, the average in 55 cases being 51.618. The average of the control blood-counts made with a Thoma-Zeiss counter in the same series of cases was 5,130,248 to the cubic millimetre, so that a volume on the scale of the hæmatokrit corresponds to 99,390 red blood-cells. Where the volume of the reds was 45 to 65, the whites occupied about one volume; in a case of leukæmia the whites occupied thirteen volumes. Of course one volume of whites corresponds to many fewer cells than a volume of reds. The examination takes only about ten minutes, and thus there is a saving of much time and labor. There are several objections which may be raised to the use of the instrument, particularly in the study of the grave anæmias, in which the volume of macrocytes varies materially from the volume of the ordinary blood-corpuscles; but there seems little doubt that when certain improvements already suggested have been made the instrument will be generally adopted.

The enumeration of the blood-plaques has so far served no distinct practical purpose. Normally there are present from 200,000 to 500,000 in the cubic millimetre.

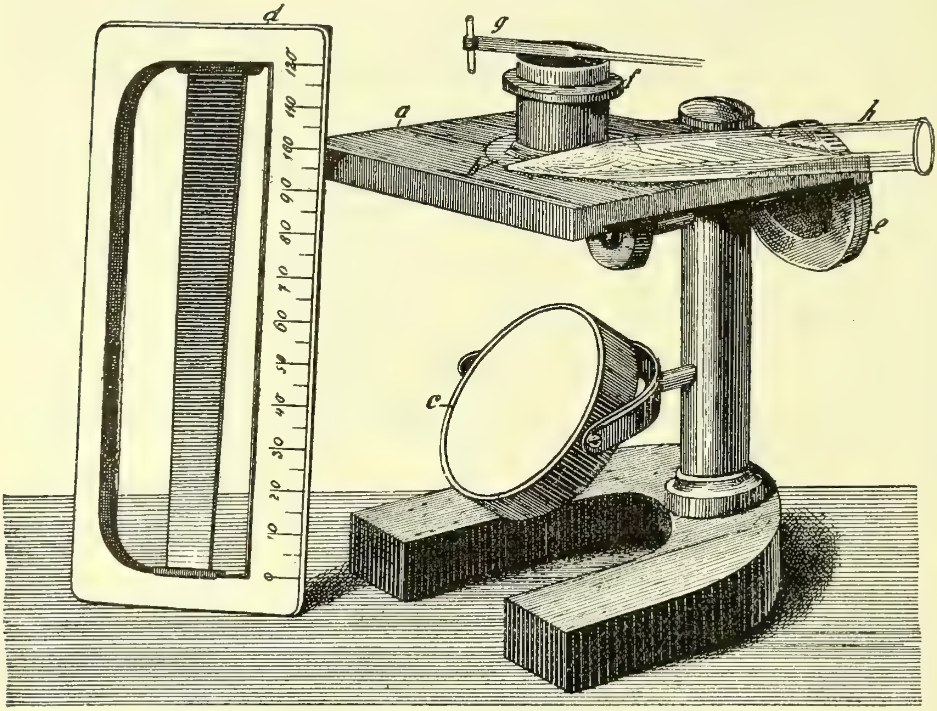
Examination of the Coloring Matter.—The instruments of Gowers (Fig. 5), Malassez, and Hayem are being entirely replaced by the convenient hæmometer devised by von Fleischl. (See Fig. 6.) Like the blood-counter of Zeiss, there go with it many little technical points inattention to which will lead to very erroneous results. The color of the blood, diluted in definite proportions with distilled water, in one compartment is to be compared with that of water in a second adjacent compartment, beneath which, by means of a thumbscrew, a wedge of red glass (Cassius's Goldpurpur) is moved until the tints of the two chambers exactly correspond. The scale showing the percentage of hæmoglobin may then be read off. The examination must be made with the aid of artificial light and in a dark room. The blood normally contains a little less than 14 per cent. of hæmoglobin, the number 100 on the scale of von Fleischl corresponding to 13.44 per cent.

Having counted the corpuscles and determined the percentage of hæmoglobin, it is easy to calculate the individual corpuscular richness in coloring matter (valeur globulaire)—a point of much practical value in the differential diagnosis of certain forms of anæmia.

There are times, however, when it is desirable to add to a quantitative the results of a qualitative estimation of the coloring matter of the blood. This may be best done by means of the spectroscope, of which certain inexpensive forms have been invented which are suitable for clinical purposes. Thus, carbondioxide poisoning leaves traces in the blood which can be recognized by the spectroscope for weeks after the intoxication.

The Study of Dried and Stained Specimens.—In the diagnosis of blood diseases it is to Ehrlich of Berlin that we owe the increased precision obtained by a color-analysis of the formed elements of the blood. He makes use of Koch's method of preparing cover-glass specimens. A drop of blood is allowed

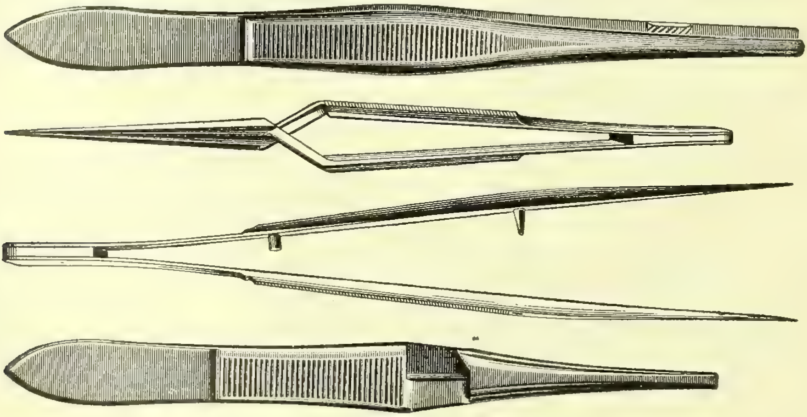
Fig. 6.



a, Stand; *b*, Opening into which the double chamber fits; *c*, White plate from which light is reflected through the chamber; *d*, Frame with the wedge of colored glass which passes under the chamber; *e*, Screw by which the glass is moved; *f*, Double chamber which holds the blood and water; *g*, Capillary tube to collect the blood; *h*, Pipette for adding the water (Limbeck).

to spread out into a thin layer between two cover-slips, which are then quickly separated and allowed to dry in the air. Forceps (see Fig. 7) are used for holding the cover-glasses, since the moisture of the fingers alters the shape of the corpuscles. Specimens so prepared may be set aside in labelled boxes and examined

Fig. 7.



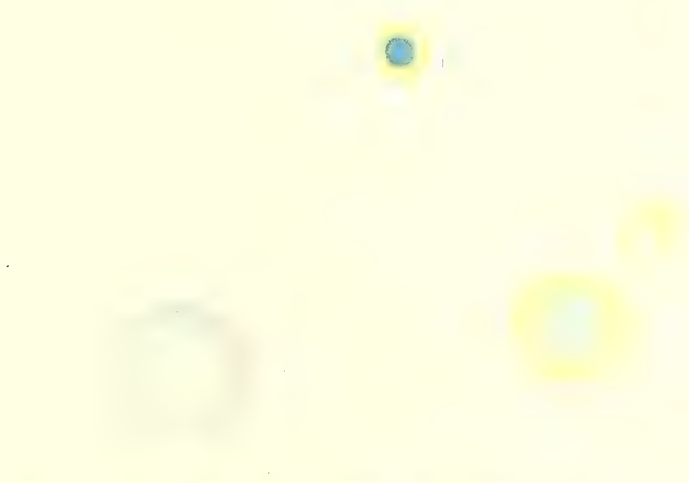
Forms of Forceps for holding Cover-glasses.

PLATE I.

FIG. 1.

Blood stained with Ehrlich's "Triple stain" of acid fuchsin, methyl green, and orange G. Drawn with the camera lucida from normal blood: *a*, red corpuscles; *b*, lymphocytes; *c*, large mononuclear leucocytes; *d*, transitional forms; *e*, neutrophilic leucocytes with polymorphous nuclei (polynuclear neutrophiles); *f*, eosinophilic leucocytes.

FIG. 2.



Same stain as in Fig. 1: *g*, myelocyte; *h*, nucleated red corpuscle (normoblast), from a case of splenomyelogenous lukæmia; *i*, nucleated red corpuscle (megaloblast), from a case of pernicious anæmia.

at leisure. Before staining the specimens must be heated (according to Ehrlich, on a copper bar) for from one to two hours at a temperature of 120° C., in order to fix the hæmoglobin of the red disks, otherwise it would be extracted by the staining fluid. In the granulated cells of the body Ehrlich finds several varieties of "specific granulations," the classification depending upon their specific behavior toward certain groups of dyes, their form, size, and conditions of solubility. He divides the aniline dyes into two groups, acid and basic—a nomenclature at first misleading, since most of the dyes are used in the form of neutral salts. By an acid dye is meant one in which the staining agent is the acid part of the salt, while in a basic coloring matter it is the base that has the staining power. A combination of a staining base with a staining acid forms the so-called neutral dye. Of these three classes, ammonium picrate might be given as an example of the first, rosanilin acetate of the second, and rosanilin picrate of the third. Altogether, Ehrlich has been able to demonstrate in the cells of different animals seven definite and distinct granulations: one cell never contains more than a single variety of granules. Only three of these are found in human blood, and only two have as yet been shown to be of practical diagnostic value:

(1) The eosinophilic, or α-granulation. (Plate I, Fig. 1 (*f*).) This is found in cells with fairly large nuclei which contain relatively large ovoid or round, highly refractive, fat-like granules, which stain by all the members of the acid group of dyes, and by no others. Erhlich has proved that these granules are made up of neither fat nor hæmoglobin, but believes them to be of an albuminous nature, a product of the secretory action of the cell-protoplasm. The granules are called eosinophilic, on account of their great affinity for eosin (tetrabrom-fluorescein).

(2) Basophilic, or β-granulation. Only rarely does one see cells in the blood whose granules stain only with color bases, though this granulation is common enough in the "mastzellen" of the tissues.

(3) The neutrophilic, or ε-granulation. This is the most important of all the granulations, and is found in the majority of leucocytes. The granules are very fine, and resolvable only with high powers of the microscope. They stain in the neutral dyes—*e. g.* in a mixture of acid fuchsin and methyl green.

Virchow in 1845 pointed out two forms of leucocytes—a large and a small. More than twenty years later Max Schultze established the fact of the absence of a morphological unity in the leucocytes, and suggested their division into groups. In normal blood we now distinguish the following varieties of white blood-corpuscles:

(*a*) *Lymphocytes.*—Small cells about the size of red blood-corpuscles, containing large, roundish, deeply-staining nuclei, the non-granular protoplasm appearing only as a little rim round the nucleus. (Plate I, Fig. 1 (*b*, *b*, *b*).) These are derived from the lymphoid tissues of the body, and make up 20 or 30 per cent. of the whole number of leucocytes in the blood.

(*b*) *Large Mononuclear Forms.*—Cells with a large oval or ovoid feebly-staining nucleus, and a relatively well-developed protoplasm which does not contain granules. (Plate I, Fig. 1 (*c*).) Ehrlich believes that they are gradually transformed in the circulating blood into the smaller polynuclear forms.

(*c*) *Leucocytes with Polymorphous Nuclei* (the so-called "polynuclear" leucocytes).—This is the most common form, since they represent two-thirds of the whole number of white blood-corpuscles. (Plate I, Fig. 1 (*e, e*).) They are smaller than the large mononuclear elements, and are characterized by the irregular forms of their nuclei, which take all sorts of shapes—S, V, Y, Z, or E. Their protoplasm is thickly studded with fine neutrophilic granules, so that they are often called "polynuclear neutrophiles."

(*d*) *Transition Forms.*—These cells are similar to the large mononuclear corpuscles, but differ in having indentations in their nuclei. (Plate I, Fig. 1 (*d, d*).) Erhlich regards them as intermediate forms between *b* and *c*. The mononuclear cells, together with these transition forms, represent about 6 per cent. of the whole number of leucocytes in normal blood.

(*e*) *Eosinophiles.*—These are cells of about the size of the polynuclear leucocytes with variable nuclear forms, and a protoplasm containing large refractive eosinophilic granules. (Plate I, Fig. 1 (*f*).) They make up from 2 to 4 per cent. of the white blood-corpuscles, and have their origin probably in the bone-marrow. Forms *b*, *c*, and *d* are said to come from both spleen and bone-marrow.

The proportions of the different forms above given are fairly constant in health, but may vary widely under different pathological conditions. Hence a "differential count" of the leucocytes may be of very great value, as I shall show later, in the clearing up of the diagnosis of a difficult case.

In some diseases cells not normally present in the blood at all may be found in dried specimens, and the presence of these, together with certain degenerative and regenerative forms of red blood-corpuscles, to be spoken of later on, are signs hitherto insufficiently appreciated.

It would be going beyond the limits of a general text-book to refer to all the various staining methods which may be advantageously used. The coloring mixture of greatest practical value is perhaps that known as the "triple stain" (Ehrlich-Biondi). It contains methyl green, acid fuchsin, and orange G. With this fluid the nuclei of the white blood-corpuscles are stained green, those of the nucleated red blood-corpuscles nearly black, the red corpuscles themselves orange, the eosinophilic granules red, the neutrophilic granules a deep violet.

The plasmodia malariæ may be studied in dried specimens stained with Plehn's solution of eosin and methylene blue, but these bodies can be examined much more satisfactorily in the fresh blood. The staining of micro-organisms in other blood infections (*e. g.* the tubercle bacillus in acute miliary tuberculosis) gives results too inconstant to be of much practical clinical value.

ACCESSORY METHODS OF EXAMINATION.

The Specific Gravity.—In health the specific gravity of the blood may vary between 1045 and 1075. It is, as a rule, higher in men than in women, and in youth than in adult life. The procedure of von Schmaltz of weighing a known volume in a capillary tube is an accurate method of determination, but takes up too much time. More convenient is the method of Hammerschlag, who places a drop of blood in a mixture of chloroform and benzol; he then adds either chloroform or benzol until the blood-drop floats lazily around in the mixture. The specific gravity of the latter is then taken in the ordinary way with an areometer. Another plan is given by Dr. Lloyd E. Jones of Cambridge.[1] The specific gravity of the blood is lowered in pulmonary phthisis, different forms of anæmia, and in certain cachexias.

The Alkalescence.—As is well known, the reaction of the blood is alkaline, but the amount of alkaline carbonates varies under different physiological and pathological conditions. The technique of the determination is complicated, and will scarcely be undertaken except for purely scientific purposes.

The Estimation of the Total Amount of Blood.—This can be at best only approximate, and need not be described here.

PLETHORA.

The older writers spoke of plethora as a definite pathological condition, depending either on an increase in the total amount of the blood in the body or on an increase of the red blood-corpuscles beyond the normal limit. They distinguished a true plethora (plethora vera) from a symptomatic plethora (plethora apocoptica, following amputation of a limb; plethora hydræmica, after hæmorrhages or in cachexias). By true plethora was meant that condition of full-bloodedness seen in men of strong constitution, where the face is generally red, the mucous membranes injected, the pulse large in volume and bounding, such patients, in consequence of their plethoric condition, suffering at times from attacks of palpitation and dyspnœa, epistaxis, and hæmorrhages from different mucous membranes. Hereditary predisposition, over-feeding, and insufficient exercise are the most important of the various causes which have been suggested. The experimental investigations of Worm-Müller and Cohnheim on animals, although they show the possibility of a temporary plethoric condition, do not justify the belief in a persistent polyæmia. Large quantities of transfused blood were disposed of in dogs in a few days, and at most in two or three weeks, after injection. The appearance of full-bloodedness in men is due rather to changes in the vaso-motor system controlling the blood-distribution than to actual increase in the total volume of blood.

The cases of blood-concentration following watery evacuations from the bowels, as in cholera, where at times there may be over six million red corpuscles to the cubic millimetre, would now scarcely be regarded as a state of plethora.

THE ANÆMIAS.

Anæmia may be a general or a local condition. The local anæmia, or ischæmia, dependent upon some obstruction to the circulation, in an individual part, will not be considered here. In a general anæmia there may be a diminution in the total amount of the blood, of its contained corpuscles, or of certain other important constituents, such as albumin and hæmoglobin. Where there is a decrease in the number of red blood-corpuscles, we speak of an oligocythæmia; when the amount of hæmoglobin is low, the term oligochromæmia is used. Very often the oligochromæmia is about proportionate to the oligocythæmia, but in other cases the relation is by no means equal; this relation is generally quoted in terms of the amount of hæmoglobin in an individual corpuscle—*la valeur globulaire* of Lepine.

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[1] *Journal of Physiology*, vol. viii.

Not every pale person has a general anæmia. There are individuals whose persistent pallor of the face is due to hereditary influences or to local vasomotor disturbances, who may have their full complement of corpuscles and of hæmoglobin.

Patients who have any advanced degree of anæmia present a characteristic set of symptoms—viz. pallor, shortness of breath on exertion, palpitations of the heart, headaches, and in women menstrual disturbances, most often amenorrhœa—symptoms which always demand a most careful blood-examination. Even with the marked improvement in the hæmatological technique of to-day it is often difficult to pass judgment on certain obscure cases, and all classifications given are at best only provisional. It will be found convenient to separate the so-called primary or essential anæmias from the secondary or symptomatic forms, it being understood that what we now call primary anæmias are so only because we are as yet unacquainted with their exact etiology.

THE PRIMARY OR ESSENTIAL ANÆMIAS.

Of these we have two distinct forms—Chlorosis, and Progressive Pernicious Anæmia.

CHLOROSIS.

Definition.—An affection occurring chiefly in young females, which produces clinically the group of symptoms common to the anæmias, and is characterized by a marked diminution of the amount of hæmoglobin in the individual corpuscles.

Etiology.—The great majority of cases occur between the ages of fourteen and twenty-four—a fact which gives some support to the view that there is an intimate relation between the affection and the changes which the organism undergoes at puberty. In girls in whom the disease occurs early in their teens we are apt to find a certain precocity and an almost premature appearance of the menses, while cases occurring later are associated with a history of a late puberty. As a rule, the pallor begins a year or two after the menses are first seen, and scanty menstruation or total amenorrhœa is a concomitant symptom, the menorrhagic chlorosis of Trousseau, as far as our experience goes, being uncommon. Girls with light hair and fair complexion are more frequently attacked than brunettes. The affection is rare in males, though some cases occurring at puberty have been reported.

Virchow has advanced the theory that a congenital hypoplasia of the vascular system lies at the bottom of the condition, and that the disease is present from birth. The affection is extremely rare in young children. Hereditary influences seem, however, to play some part in the etiology; thus a mother who has been chlorotic not infrequently bears children who later on become chlorotic, and the physician frequently has to treat two or more sisters suffering from chlorosis at the same time. There are facts which indicate that the disease occurs more frequently in families contaminated with tuberculosis (Jolly).

A primary nervous origin is claimed by some who cite instances of chlorosis developing after sudden shock or violent emotion, homesickness, or disappointment in love. The "green and yellow melancholy" of Shakespeare was probably a poetic term for this disease. The influences of unsatisfied sexual desires and of masturbation have, it seems to me, been over-estimated. In dispensary practice a large proportion of the cases are found among hard-working sewing-girls or factory-operatives, who have long hours, eat in haste improperly prepared food, work in close, ill-ventilated, and badly-lighted rooms, and have several flights of stairs to climb every day. Such a constant transgression of all hygienic laws in regard to air, food, and exercise cannot fail to exact its penalty from the constitutions of young girls, already severely taxed by the assumption of the functions of womanhood. The better classes are, however, not exempt, and the disease is frequently found in the most luxurious homes. Sir Andrew Clark found constipation so frequently in his cases that he regards the affection as the result of the absorption of toxic products from the colon—a true copræmia.

Morbid Anatomy.—The pathology of chlorosis is imperfectly understood. Few cases die directly from the disease, and the pathological findings in those which have come to autopsy have been by no means constant. Rokitansky in 1846 pointed out certain instances of incurable chlorosis due to anomalies of the blood-vessels and of the genital organs. Virchow described a congenital hypoplasia of the vascular system found in several autopsies on chlorotic patients. The aorta and all its branches were of small calibre and thinner than normal; the elasticity of the vessels, however, appeared to be increased. In some of his cases, but by no means in all, the genital organs also showed errors in development. The heart is at times dilated and the left ventricle hypertrophied.

Symptomatology.—The symptoms may appear with comparative suddenness—a young girl apparently in blooming health reaching an extreme degree of pallor in a few weeks—but this is not the rule, the onset in the majority of cases being gradual. Headache is common in the early stages, and dyspnœa and palpitation are not often absent, the patient complaining of these rather than of the pallor when she consults the physician for the first time. More often still an anxious mother brings a daughter whose menses have ceased, and the physician is begged to direct his treatment toward a restoration of this function.

The general symptoms of chlorosis are those of an anæmia of a moderate grade. The patient is generally well nourished, and the panniculus adiposus rather increased than diminished. The skin in many cases has a characteristic greenish-yellow tinge, quite different from the blanched aspect produced by hæmorrhage or the muddy pallor of the graver forms of anæmia. It is this curious tinge of the complexion which has given rise to the popular name of "green sickness:" it must be borne in mind, however, that in many patients undoubtedly chlorotic this sign may not be marked. Aside from the hæmometric examination, the degree of hæmoglobin-poverty may be judged best by examining the color of the palpebral conjunctivæ and that of the matrix of the finger-nails. The lips and cheeks may be of a rosy-red color on exertion, even when the *valeur globulaire* is considerably diminished (chlorosis rubra). The skin about the joints not infrequently shows areas of pigmentation. The breathlessness, palpitation, and tendency to fainting are evidences of the insufficient oxygenating power of the circulating blood.

The symptoms referable to the digestive system are often interesting. In some cases the functional disturbances are so marked that many authors speak of a "dyspeptic type" of chlorosis. The appetite is poor in many patients—more often it is perverted, the girl refusing to eat ordinary articles of diet, but longing for unwholesome dishes like pickles or other highly-flavored foods. School-girls have been known to eat quantities of the most indigestible substances, such as bits of chalk, slate-pencil, or even earth. Vomiting, or rather regurgitation of food, in the mornings is not uncommon. More than once patients have come to me fearing that they were pregnant—a fear perhaps not unnatural in presence of the three conditions of exposure, amenorrhœa, and morning vomiting. Contourier has found a dilated stomach in many chlorotics, and believes that it is sometimes a cause, sometimes a result, of the disease. Pick of Prague goes so far as to attribute chlorosis to the absorption of toxic products from the stomach, and claims to have successfully treated many of his cases by systematic lavage.

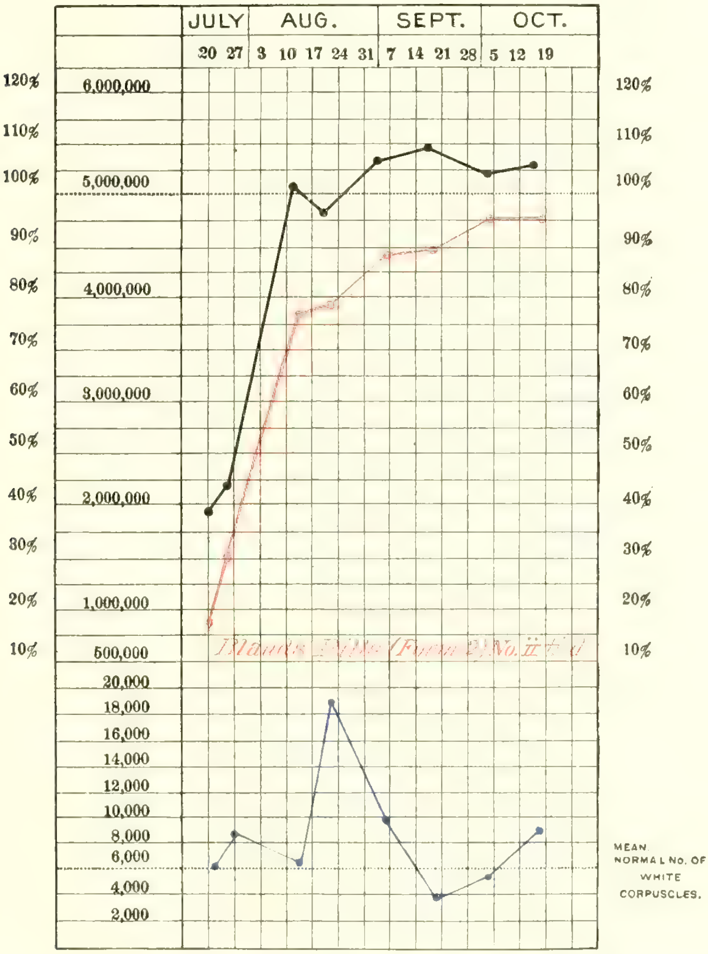
We have already mentioned the frequency of constipation in chlorosis, as well as disturbances in the menstrual function, which are extremely common. The amenorrhœa, leucorrhœa, and dysmenorrhœa generally disappear rapidly under treatment directed toward the defective condition of the blood. Nervous symptoms are more prominent in some cases than in others. Headache, particularly the vertical variety, is especially often met with, while neuralgia and even hysterical manifestations sometimes occur. Fever is not common, but there are cases (febrile chlorosis) in which there is a daily rise of temperature.

The changes in the vascular system are more or less characteristic, and the palpitation of the heart is at times most troublesome. On physical examination the heart may be found slightly enlarged, and murmurs may nearly always be heard in the severer cases, the most common being the systolic souffle at the base, usually in the pulmonary region. More rarely a whiff with the first sound is to be heard at the apex. Duroziez affirms stoutly that these murmurs may always be distinguished from the murmurs of organic disease. He claims that while the latter are still plainly heard when the ear is removed a little distance from the chest, the former under the same circumstances disappear. A loud hum, the so-called "bruit de diable," is often heard in the cervical veins. The importance of venous thrombosis in chlorotic patients has been emphasized by Brayton Ball. It may occur in the femoral or brachial veins, where it is not apt to be serious, but when the longitudinal sinus is thrombosed there may be a fatal result.

Œdema of the ankles sometimes occurs, but it is not so common here as in the graver anæmias.

The Blood Examination.—The red globules may be present in normal numbers, although in all severe cases there is a considerable oligocythæmia.

Fig. 8.



Blood-chart of Case of Chlorosis: black, red corpuscles; red, hæmoglobin; blue, colorless corpuscles.

In Thayer's series of 63 cases, studied in my clinic, the average number of red blood-corpuscles to the cubic millimetre was 4,096,544, or 74 per cent. of the normal, and the average quantity of hæmoglobin was 42.3 per cent. This relatively great oligochromæmia, first pointed out by Duncan, is the distinguishing characteristic of the blood in chlorosis, and serves to differentiate it sharply from diseases, like pernicious anæmia, where the globular value in hæmoglobin is increased. In one case the oligocythæmia was severe, there being only 1,953,000 reds, with 17.5 per cent. of hæmoglobin. The white cells were only slightly increased in number, averaging in the above 63 cases 8467 to the cubic millimetre—*i. e.* 1 white to 408 red. One case with over 85 per cent. of red globules and only 35 per cent. of hæmoglobin presented accurately the clinical aspect of a profound anæmia.

If a drop of fresh blood be examined, the pallor of the individual corpuscles is at once apparent. There may be many poikilocytes, by which we mean deformed red globules assuming the most curious flask-like, hammer-shaped, or pyriform appearances. In the dried and stained specimens a small nucleated red blood-corpuscle (the normoblast of Ehrlich) may now and then be seen. Græber's determinations, according to Landois's procedure, showed a diminution in the alkalinity of the blood, but investigations by more accurate methods have not confirmed this.

Diagnosis and Prognosis.—When a young girl comes to us with such an array of symptoms and complaints, one should never assume the responsibility of excluding a grave anæmia before making a careful blood examination, although the diagnosis in the majority of cases is tolerably easy. There are cases of secondary anæmia, however, with considerable diminution in the corpuscular hæmoglobin value : this is especially true of the anæmia of the earliest stages of tuberculosis of the lungs. One should always satisfy himself that the lungs are clear before giving a positive diagnosis, and organic diseases of the heart and kidneys are to be excluded in the ordinary way.

The prognosis is always favorable, except in those cases associated with congenital anomalies of the vascular and genital systems. We must not, however, forget that relapses are common, and where they occur it is in most cases because the treatment has been too soon discontinued. It is not rare to see a recurrence during the third decade of life.

Treatment.—Chlorosis is one of the few diseases of which the physician is a therapeutic master. A few weeks' administration of iron, together with an improved hygienic condition, usually suffice to restore a ruddy glow to the most pallid cheek. At the outset the patient should be impressed with the desirability of persevering with the treatment until the hæmoglobin value, *as shown by the hæmometric scale,* is above 90 per cent. The distressing symptoms may disappear entirely at the end of two weeks—long before the percentage of the hæmoglobin is normal—and thus our patient may disappear for weeks, only to return in her former condition. In the severest case on Thayer's list, where the red blood-corpuscles were below two millions and the hæmoglobin below 20 per cent., at the end of four weeks the numbers of reds were found to have increased to 5,090,000, and the hæmoglobin to 70 per cent., and at the end of the eleventh week her hæmoglobin percentage was 95. All of the cases of the series were given iron in the form of Blaud's pills, two pills of five grains each after every meal. The patient was encouraged to spend one or two hours in the open air daily, and to take three or four glasses of milk between her meals in the course of the twenty-four hours. Where necessary, constipation was treated by salines in the morning.

If one form of iron disagree, we shall often find that another preparation may be taken without trouble. How the iron acts is still unsettled, but the theory of Bunge is a very plausible one. He believes that inorganic iron given in any form is not itself absorbed, since an equal amount can always be detected in the fæces, but supposes, on the other hand, that it unites with the hydrogen sulphide or other sulphur compounds in the gastro-intestinal tract, and thus permits the absorption of the highly complex organic combination of iron which exists in food-stuffs, and which would otherwise pass off in the fæces as an insoluble sulphide. Certain ingenious pharmacists, influenced by this hypothesis, have put upon the market preparations of albuminate of iron, but these are all of doubtful advantage. The diet should be liberal and nutritious.

As the girl gradually regains her color, acneiform eruptions on the face are not uncommon, but these are of trifling importance and yield to the ordinary treatment. I have found the systematic examination of the blood with v. Fleischl's instrument a valuable aid in encouraging patients to continue treatment until cured, as they grow interested in watching the steady increase in the hæmoglobin percentage.

PROGRESSIVE PERNICIOUS ANÆMIA.

This disease was first clearly described under the name of "idiopathic anæmia" by Addison, whose account of the clinical history of the affection has become classical. Wilks, Lebert, Channing, and Gusserow all have added to the literature of the subject. Thus far, the disease had been studied chiefly in its clinical aspects, and it is to Biermer, who in 1868, discussed the pathology of the affection, that we owe the revival of interest in the affection, which since then has been studied by a host of observers.

Etiology.—Addison as early as 1843 had spoken of the disease in his clinics; and in his monograph on the suprarenal capsules published in 1855, we find a brief account of this form of anæmia, of which he speaks as follows: "For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever—cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease." The name "essential anæmia" had its origin with Lebert of Zurich (1858), while the term "progressive pernicious anæmia" was coined by Biermer, who thought he was dealing with a previously unknown affection.

Addison's anæmia is geographically widely distributed. It was thought at one time to be particularly common in the cantons of Switzerland, owing probably to the fact that it was confused with some of the many grave cases of secondary anæmia seen there. Many cases have been described in this country, and twenty-eight have come under my personal observation. Although the disease sometimes occurs in children, it is much more common in those of middle age. The youngest patient I have seen was a girl of twenty, but Griffith has collected some ten cases in patients under twelve years of age. Males are attacked more frequently than females.

There are, associated with certain known conditions, many instances of progressive and fatal anæmia which cannot be distinguished clinically from the idiopathic form of Addison. Severe anæmias accompanying pregnancy and parturition make up a large proportion of the reported cases of pernicious anæmia. Again, certain atrophic conditions of the gastric mucous membrane give rise to an anæmia at once progressive and pernicious, but by means of the improved technique for the investigation of the gastric contents it will sometimes be possible to exclude these *intra vitam.* Lastly, the grave secondary anæmias consequent upon the presence of intestinal parasites (Anchylostoma duodenale, Bothriocephalus latus) may be extremely difficult to recognize until after death.

In eighteen of my cases, however, there was absolutely no appreciable cause for the anæmia, and they therefore correspond to Addison's description. As yet we are unable to say definitely whether the cause of these obscure anæmias is to be looked for in a hæmolytic process or in a defective hæmogenesis. Stephen Mackenzie, F. P. Henry, and others believe that, owing to some fault in the process of blood-making, the corpuscles become abnormally vulnerable. The weight of opinion, however, on the whole, is in favor of an increased hæmolysis. The experiments of Quincke and Peter with regard to the enormous increase of iron in the liver, and those of Hunter bearing on the excretion in the urine of quantities of pathological urobilin, are interesting in this connection.

Birch-Hirschfeld holds that the tissue-destruction and the retardation of blood-coagulation favor the idea of an infectious origin. Unfortunately, our knowledge of the etiology is as yet far too limited to enable us to construct any theory which is wholly adequate or satisfactory. Only by the most careful and minute examination after death of patients whose blood during life has been carefully studied according to modern methods can we hope to find the solution of the problem.

Morbid Anatomy.—The pallor of the whole body surface and of the organs is striking, and a characteristic lemon-yellow tint of the skin is present in many cases. As a rule, the body is not emaciated, and, as in most anæmias, the subcutaneous fat is not diminished. The muscles may be pale, but are often intensely red. Punctiform hæmorrhages in the organs and on the serous membranes are quite common. The lungs are not particularly abnormal. The heart-muscle is very pale, light yellow in color, and shows in fresh teased preparations the most intense fatty degeneration. The walls of the ventricles are remarkably lax and flaccid, and the cavities contain light-colored blood. The intima of many of the smaller vessels may show patches of fatty degeneration. The stomach in the purely idiopathic cases is normal, except for slight fatty degenerative changes in the cells of the secreting tubules. The anæmia resulting from extreme atrophy of the mucosa must of course be no longer regarded as primary. The liver in most of my autopsies was normal in size and fatty. In some cases it was enlarged. The peculiar distribution of the iron in the liver seems to be characteristic of these cases, the pigment being deposited in the outer and middle zones of the lobules, and in two of my specimens appearing to outline the bile-capillaries. This is quite a different picture from that seen in secondary anæmia, and may possibly be peculiar to the disease. The liver in forty-five consecutive autopsies examined for me by A. C. Scott showed this special lesion in no cases other than those of pernicious anæmia.

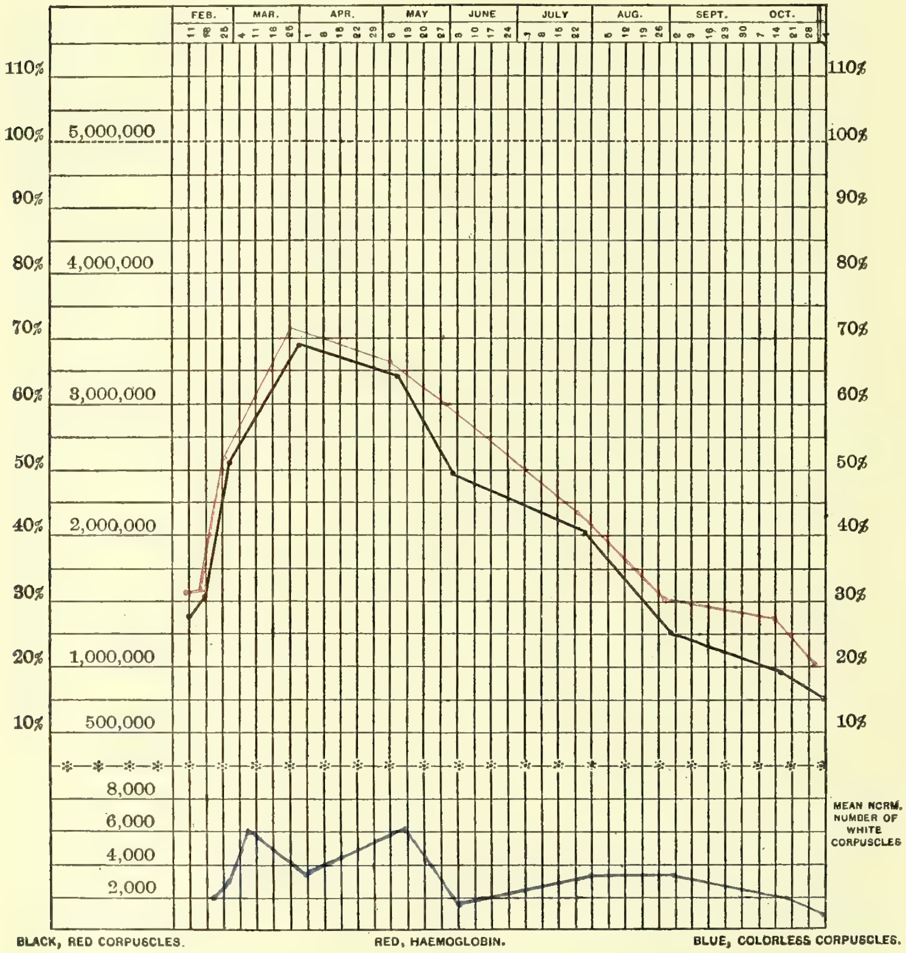
Naturally, the hæmopoietic organs have been the objects of anxious study. The spleen shows no characteristic lesions; the amount of iron in it is usually increased. The lymph-glands may be unchanged, though in three of my cases they were of a rich deep-red color resembling spleen-tissue—a condition which has also been noted by Weigert. The amount of yellow marrow is diminished, and is apparently replaced by hæmoblastic red marrow. In a case reported by Rindfleisch the marrow appeared to be one huge mass of nucleated red cells, and Rindfleisch is inclined to think that the cause of the pernicious anæmia was an inability of the organism to change the nucleated red cells into the normal non-nucleated red blood-corpuscles.

Certain other lesions have been described, but none of them are constant. Such are the changes in the sympathetic ganglia mentioned by Queckett, Wilks, and Brigidi, and the sclerosis in the posterior columns of the spinal cord noted by Lichtheim.

Symptomatology.—An individual who perhaps has before been perfectly healthy begins gradually to develop the symptoms of an anæmia. Occasionally the onset is rapid, but as a rule it is so insidious that the patient is scarcely able to give the exact date of the beginning of his illness. He gradually becomes paler, and notices that he tires easily, gets out of breath, and has palpitation of the heart on the least exertion. Headache, vertigo, and ringing in the ears are not uncommon as early symptoms. All these may be gradually aggravated, and later on the pallor may be extreme, the skin assuming the characteristic lemon-yellow tint. The digestive organs become disturbed; the appetite is poor, and nausea and vomiting are frequent. The ankles become œdematous, and hæmorrhages may take place into the mucous membranes. The end is graphically described by Addison: "The debility becomes extreme, the patient can no longer rise from bed, the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

THE BLOOD EXAMINATION.—The oligocythæmia is always marked, and is generally extreme; in one case reported by Quincke there were only 143,000 red corpuscles to the cubic millimetre just before death. The oligochromæmia does not keep pace with the cell-reduction, the percentage of hæmoglobin always being higher relatively than the percentage of red globules. In one of my cases the hæmoglobin percentage was greater by 10 per cent. This relative increase in the individual globular richness is an important point in the differential diagnosis between this disease and chlorosis, as well as the secondary anæmias. The value of this sign was first noted by Laache, and it has by many been considered pathognomonic of the disease.

Fig. 9.

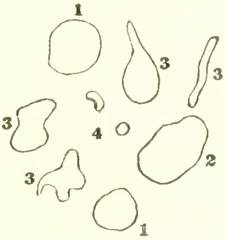


Blood-chart of Case of Pernicious Anæmia.

While we acknowledge and appreciate its diagnostic importance, yet we must warn the student that even with the best clinical hæmoglobinimeter (v. Fleischl's) which we possess the determination of the amount of coloring matter can only be approximate. Although in blood which is nearly normal the error may not amount to more than 2 per cent., Neubert and Letzius[1] have shown that when one examines a much-impoverished blood, such as is always found in pernicious anæmia, the error may be as great as 20 per cent., or one-fifth of the number read on the scale. Since the difference in relation between corpuscular and hæmoglobin percentages will never be very great, one would always be left more or less in doubt as to whether there were an actual increase in the *valeur globulaire* or not.

The fresh blood-slide reveals many megalocytes or macrocytes (Fig. 10 (2).)—large red globules measuring from ten to fifteen micro-millimetres across —which have been spoken of by Henry as indications of a "reversion to a lower type." They are a constant feature in the disease, and it is supposed to be their large size which accounts for the relative hæmoglobin-increase. Furbringer, basing his opinion on a study of seventy-five cases, claims that only those anæmias are to be regarded as pernicious where one-quarter of the red blood-corpuscles are macrocytes. Besides these, there are many dwarf forms known as microcytes, (Fig. 10 (4).) first described by Vanlair and Masius, which measure from 2 to 6 μ in diameter, as well as misshapen poikilocytic cells (Fig. 10 (3).) which are very frequent. There is no leucocytosis; indeed, the number of leucocytes would even appear to be diminished. The blood-plates are few in number or absent altogether.

Fig. 10.



Red corpuscles from a case of profound anæmia. 1, 1. Normal corpuscles. 2. Large red corpuscle—megalocyte. 3, 3, 3. Very irregular forms—poikilocytes. 4. Very small, deep red corpuscles—microcytes.

One turns with interest to the results obtained from the study of dried and stained specimens. There is nothing remarkable about the leucocytes, except that the large mononuclear elements are relatively somewhat increased. Ehrlich, however, has pointed out the constant presence of two varieties of nucleated red blood-corpuscles in the disease: (1) the ordinary form or normoblast, which is about the size of an ordinary red globule, and contains a nucleus which stains intensely and is always placed excentrically in the cell; and (2) very large forms with large faintly-stained nuclei (megaloblasts or gigantoblasts of Ehrlich). (Plate I, Fig. 2 (*h*) and (*i*).) The former correspond to the nucleated globules occurring in the blood-forming organs in adult life, the latter to those of embryonic blood-development.

The megaloblasts are found only in very small numbers, and then much degenerated, in the secondary anæmias; in pernicious anæmia and in the later stages of leukæmia they are numerous. Ehrlich's description of the different forms of degeneration of the red disks will be found of great interest. More particularly we would draw attention to the retrogressive changes revealed in specimens stained doubly with hæmatoxylin and eosin.

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[1] *Inaug. Diss.*, Dorpat, 1889.

THE CIRCULATORY SYSTEM.—The important symptoms referable to the cardio-vascular system have been already mentioned, of which the distressing palpitation may give the patient most concern. On physical examination, besides the loud venous hum in the neck, murmurs may always be heard over the cardiac area. There may be visible pulsation and throbbing of the larger arteries, and in two of my cases venous pulsation was noted. A capillary pulse is frequently to be seen, and a sphygmographic tracing of the collapsing pulse may be quite suggestive of aortic insufficiency. Hæmorrhages into the skin and mucous membranes are by no means rare, and there may be retinal hæmorrhages, causing blindness or partial limitation of the visual field. An ophthalmoscopic examination should be made in all severe cases of anæmia. The tendency to fatal thrombosis spoken of in connection with chlorosis is never seen in pernicious anæmia.

THE RESPIRATORY SYSTEM.—In the earlier stages dyspnœa is commonly present—a symptom which later may be much aggravated, even to such an extent that the breathing becomes stertorous (anæmic dyspnœa). Toward the end œdema of the lungs and dropsical effusions may be looked for.

THE DIGESTIVE SYSTEM.—The lips and tongue are pale; the appetite is variable, but is generally poor. Dyspepsia, nausea, vomiting, and diarrhœa may be present throughout the whole course. The liver, as a rule, is of normal size, but the spleen is often slightly enlarged and its border at times palpable.

The urine gives evidence of decided changes in tissue-metamorphosis. The urea and uric acid are increased. A low specific gravity along with a dark-colored urine is more or less characteristic. Hunter and Mott, who have investigated the urine chemically, have proved this to be due to the presence of pathological urobilin, a substance differing in many ways from the urobilin found in normal urine. The addition to the urine of a few drops of an alcoholic solution of zinc chloride gives a marked green fluorescence, but the substance is best detected by spectroscopic examination. If pathological urobilin be present, a well-marked absorption-band will be visible lying close to the line *F* and fading off toward *b*, with a considerable absorption of the outer part of the blue spectrum. Peptonuria has little or no significance.

Fever may or may not be present. Thus, a normal temperature may be noted for weeks, and afterward be followed by an irregular pyrexia. The increased sensitiveness of certain bones (*e. g.* the sternum) to pressure has probably been exaggerated. With the "anæmic sclerosis" of the cord there are in some cases disturbances of sensation, and in one instance an extensive paralysis was observed by Lépine.

Diagnosis.—The essential points to be noted are—(1) the severe grade of oligocythæmia; (2) the increased *valeur globulaire;* (3) the presence of many macrocytes and gigantoblasts; (4) the absence of any cause for secondary anæmia; (5) occasional febrile disturbances; (6) the yellow tint of the skin; (7) hæmorrhages, particularly retinal; (8) a progressive course and the inefficiency of treatment.

Pernicious anæmia may be readily distinguished from chlorosis by the clinical examination of the blood, which will reveal in the former disease the increased globular richness in hæmoglobin and the presence of Ehrlich's gigantoblasts. Again, as we have said, the oligocythæmia in chlorosis is never very marked. The differential diagnosis from some of the severe forms of secondary anæmia may be extremely difficult. One can of course in most cases attain to a moderate amount of certainty from the study of the blood. The origin of the grave secondary anæmias associated with gastric cancer or atrophy of the gastric mucosa may perhaps be cleared up by examining the stomach-juice according to modern methods. The skin, as a rule, too, in these cases has not the yellow tint, although the patient may present the signs of severe cachexia. Where intestinal parasites are suspected and the patient's condition will permit, a brisk purge may be given and the fæces examined microscopically for the parasites or their eggs. Secondary anæmia consequent on pulmonary tuberculosis or renal disease will scarcely be overlooked by a well-trained clinician, but even the shrewdest diagnostician will sometimes be able to discover at the autopsy some cause for a secondary anæmia which he has regarded during life as an anæmia of the true Addisonian type.

Prognosis.—The prognosis in a majority of cases is, as we might expect, very grave. Up to a short time ago the disease was supposed to be invariably fatal; indeed, some authors even now look upon reported cases of cure as examples of mistaken diagnosis. Since the introduction, however, by Byrom Bramwell in 1877 of the arsenic treatment, the results have been more favorable. Some cases appear to have been entirely cured, and in many the progress of the affection, at least for a time, has received a decided check. Of my own series of 28 cases, 2 have recovered under Fowler's solution; 16 are dead; several of the others remained in comparatively good health for a few years, and disappeared from observation. Relapses after marked improvement are extremely common, and such an occurrence is so characteristic that Stephen Mackenzie in his recent lectures makes a distinct class of what he calls cases of "relapsing pernicious anæmia." I myself know of no instance in a male in which the improvement was maintained for more than five years, but one case reported recently by Hale White has enjoyed good health for eleven years after cure by arsenic.

Treatment.—In contradistinction to the beneficial effects of iron in chlorosis, in this disease the drug seems to do little or no good, and it is on arsenic that we must mainly rely. It may be given in the form of Fowler's solution in gradually increasing doses, but if the liquid preparation be not well borne, the patient will probably do well on pills of arsenious acid. My plan is to start with 3 minims of Fowler's solution after each meal, and increase to 5 at the end of the first week, to 10 at the end of the second week, and so on until the patient is taking from 20 to 25 minims thrice daily. In one of my cases which remained well for three years I was able to push the drug up to 30 minims at a dose. The patients occasionally do surprisingly well, and toxic effects from the drug are unusual. Some persons, though, are more susceptible than others, and as soon as some œdema of the eyelids or digestive disturbances are noted, the administration of the drug should be discontinued until all such symptoms have disappeared, and then resumed at the dose at which we left off.

Rest in bed is essential at the beginning of the treatment. A light but nutritious diet is highly desirable, for the longer the digestive powers hold out the greater the hope. As a rule, the cases are best treated at home, removal to the seaside or mountain resorts being often disappointing. Birch-Hirschfeld[1] recommends, especially in the early stages, a residence in some immune place, citing Munich as an example. A systematic massage treatment is often of great benefit. The use of rectal injections of dried blood I can no longer advise. In the later stages the question of blood-transfusion arises: it is of doubtful advantage, and is certainly not free from danger. If anything be used, I would recommend the introduction of a warm physiological salt solution into the subcutaneous tissues, but even this can at best give only temporary relief.

The Secondary Anæmias.

This includes all those cases of anæmia resulting from hæmorrhage and those coming on in the course of other affections. The blood-impoverishment is here due to a definite cause, and the consequent anæmia is, as a rule, directly proportionate to the severity of the primary affection. But why under similar conditions, with apparently the same etiological factors at work, the anæmia in one case may be slight and in another profound is difficult to explain.

The Blood.—No matter what the cause, the blood in the symptomatic anæmias presents certain characteristics which are more or less constant. The degree of oligocythæmia may vary from a slight diminution in the number of corpuscles in a mild case to an enormous decrease, almost as great as would be seen in a case of pernicious anæmia. The amount of hæmoglobin decreases *pari passu* with the number of red blood-corpuscles. At times the individual globular richness may be below par, but there is never an increase, such as occurs in pernicious anæmia. The number of white blood-corpuscles is always relatively, and generally absolutely, increased.

The fresh blood-slide varies in its appearances according to the degree of anæmia. In mild cases little or nothing abnormal may be noted, while in the severer grades one will find as marked alterations in the size and shape of the corpuscles as are ever seen in one of the essential anæmias. Microcytes, macrocytes, and poikilocytes in such a case will be numerous. In stained specimens nucleated red blood-corpuscles can always be found, although the search may be a long one if the anæmia be slight. It is the normoblast that predominates here, the megaloblasts being rarely seen. If there be a leucocytosis, the extra leucocytes are almost always polynuclear neutrophiles. This increase, which is generally present, is most marked in the anæmia following a severe hæmorrhage.

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[1] *Deut. med. Woch.*, 1892, Apr. 1, 28.

Classification of the Secondary Anæmias.

Any arrangement of the secondary anæmias into groups has thus far been unsatisfactory, having been of necessity based on the etiology. Given a case of secondary anæmia, it may often be difficult to decide which one of several possible causal agents present may have been responsible for the blood-impoverishment.

The most important groups are—

1. Anæmia from Hæmorrhage.—The loss of blood may be the result of a lesion of a large vessel from injury or from rupture of an aneurism or from post-partum bleeding, or there may be severe hæmorrhage in cases of ulcer of the stomach or duodenum, or in cirrhosis of the liver from the œsophageal varices, and in various other conditions. By the so-called spontaneous hæmorrhages we mean those occurring in individuals suffering from a hæmorrhagic diathesis—*e. g.* in purpura, scurvy, and hæmophilia. Where the bleeding takes place quickly there is a diminution of all the blood-constituents, a true oligæmia. The total volume of blood may be so much diminished and the general arterial tension so lowered that death results in a few moments, and the more sudden and profuse the loss the greater the danger of fatal syncope. Thus, the rapid shedding of three or four pounds of blood would probably be fatal. In one case which I saw seven and a half pounds of blood, an unusually large amount, escaped into the pleura from rupture of an aneurism. On the other hand, where there are frequent hæmorrhages, provided always the blood-loss at any one time is small, an almost incredible amount may escape and yet the patient ultimately recover. Thus, I have known a man to lose over ten pounds of blood in one week from gastric hæmorrhage without succumbing.

Sometimes we may be unable to obtain a history of hæmorrhage, as in cases of enterorrhagia from the anchylostoma duodenale, bothriocephalus latus, or from cirrhosis of the liver, where the patient himself may be unaware of any such loss, and where for some reason or other the history is withheld, as may happen in the cases of female patients suffering from bleeding piles or metrorrhagia.

It is surprising to find how rapidly the regenerative processes go on, the normal amount being regained sometimes in a week or ten days after the hæmorrhage if it occur in a previously healthy individual. The lowering of the arterial pressure permits the absorption of lymph from the perivascular spaces in the tissues, even while the hæmorrhage is still going on. The albuminous, watery, and saline constituents are much more quickly renewed than the cellular elements, and it may be even weeks or months before there is a complete *restitutio ad integrum.* The restoration of the hæmoglobin does not keep pace during this regeneration with the corpuscular development.

2. Anæmia from Inanition.—Here the plasma, as a rule, suffers more than the corpuscles; the latter may be present in almost normal numbers to the cubic millimetre. The inanition may be due either to a deficiency in the food-supply or to disturbances of one or more of the various physiological processes by which digestion and absorption are carried on. Good examples of this form of anæmia are seen in cancer of the œsophagus or pyloric orifice, and in the chronic dyspepsias, especially in those due to atrophy of the gastric mucous membrane.

3. A prolonged drain on the albuminous materials of the blood, no matter what the cause, is accountable for a large group of cases of anæmia. The blood-impoverishment of Bright's disease, of chronic suppuration, of prolonged lactation, etc. may be explained in this way.

4. Toxic Anæmia.—Under this heading may be tabulated the anæmias resulting from various poisons, organic as well as inorganic. The influence of lead, arsenic, mercury, and phosphorus as blood-globule destroyers is well known. Of the infectious diseases, tuberculosis, syphilis, and malaria are most frequently associated with anæmia. In malarial blood one may watch the corpuscles grow pale under the action of the plasmodial parasite. A post-typhoid anæmia is not uncommon, and in some cases the grade may be severe. In these toxanæmias the red blood-corpuscles may be directly destroyed as in malaria, or the ordinary rate of their consumption may be increased.

The anæmia accompanying pyrexia is due partly to the direct action of the poison producing the fever on the blood itself, partly, perhaps, to some interference with the working of the hæmopoietic organs.

Treatment.—Obviously, the first indication in the treatment will be for the removal of the cause. In the secondary anæmias, as in other secondary affections, this is unfortunately too often impossible. A careful physical examination of the organs and an examination of the gastric juice, sputum, urine, and fæces by modern methods will often reveal the primary cause. It might seem unnecessary to speak of this were it not well known that the anæmia accompanying malaria has been treated with iron before any attempt was made to kill off the swarms of plasmodia which were the active cause of the blood-degeneration. In every case strict attention must be given to the dietetic and hygienic management, a part of the treatment fully as important as the administration of drugs. The blood in the anæmia following sudden hæmorrhage in individuals previously healthy may be restored with astonishing rapidity, and often without any medicine.

It is generally best to begin by confining the patient to bed. For the anæmia, apart from the treatment of the primary cause, iron is the best drug. The particular form chosen must depend in each case to a great extent on the idiosyncrasies of the patient and the condition of the alimentary tract. In by far the greater number of cases Blaud's pills were found to be perfectly satisfactory, though some patients do better on the officinal tincture of the perchloride. Janeway prefers the tartrate of iron and potassium, but perhaps it may be necessary to search still further before the preparation appropriate to the case in hand will be found. The waters of the various chalybeate springs will sometimes prove efficient. Still, where iron is not well borne, we must fall back on arsenic.

The Leucocytoses.

Besides the different forms of leukæmia to be presently discussed, there are many conditions in which the number of leucocytes in the blood is increased. These non-leukæmic processes since the time of Virchow have been spoken of as leucocytoses. In his *Cellular Pathology* he describes a "physiological" leucocytosis occurring after meals and in pregnancy, and a "pathological" leucocytosis belonging to acute inflammations, such as erysipelas and pneumonia. He explained the phenomenon by supposing that the substances carried to the lymphatic glands stimulated their cells and caused them to proliferate, and, as the leucocytes were thought to arise from a proliferation of these cells, it was readily conceived that they should immediately be found in greater numbers in the blood. Since then numerous observers have studied the blood in the most varied diseases, and found in many, particularly in the acute suppurative diseases, a pronounced leucocytosis.

Much light has been thrown on the subject since the discovery of the existence of chemotactic processes. When one considers the tremendous number of leucocytes which accumulate in a short time in the formation of a large abscess, he cannot help wondering whence they have all arisen. Inasmuch as in all acute suppurative processes there is an extensive increase in the number of the leucocytes in the blood, besides the aggregation of leucocytes at the seat of inflammation, it is obvious that the leucocyte-building organs are capable of being suddenly aroused into an enormously increased activity. The study of pus and of dried and stained specimens of the blood from these cases shows that the extra number of leucocytes is made up almost entirely of polynuclear neutrophiles.

Speaking generally, we are able to guess in any acute disease whether or not there will be a leucocytosis. If the disease be one in which there is a pronounced local reaction—*i. e.* a disease associated with inflammatory exudation in a certain part of the body—then there will almost certainly be an increase in the number of polynuclear neutrophiles also in the blood. On the other hand, where there is little or no local reaction, no matter how intense the general process, then we shall expect only a slight leucocytosis or none at all.

The local reaction is to be regarded as the result of a positive chemotaxis. There are, as we now know, certain substances which attract, and certain others which repel, the white blood-corpuscles. Such substances are spoken of respectively as being positively and negatively chemotactic. Of their nature we as yet know but little; it seems probable, however, that they are products closely allied to the alkali-albuminates which result from the necrosis of certain tissue-cells. Buchner and his pupils conclude from their investigations that the protein substances of the bodies of micro-organisms are positively chemotactic, and that the presence of dead bacteria suffices to account for the attraction of the leucocytes (Römer). This does not, however, explain the chemotaxis resulting from the injection of substances like turpentine, nor that which taken place about certain necrotic areas in the liver and lymph-glands which have been shown to bear no direct relation to micro-organisms. The subject, however, is too wide to admit of a full discussion here. We would emphasize the fact that leucocytosis occurs, as a rule, only in diseases which have a local reaction, and that its extent is proportionate to the latter, so that we are able to say *a priori* in a given infectious disease whether or not a leucocytosis will exist. Thus in a croupous pneumonia or in a suppurative pleurisy there will be a leucocytosis proportionate to the extent of the lung or pleura involved, while in typhoid fever or malaria, where there is no marked local reaction, there will be little or no leucocytosis. Indeed, in typhoid the number of leucocytes would appear to be diminished. This peculiar character of the blood in typhoid fever furnishes us with a ready method of discovering complications in that disease. I have often in my own wards seen a leucocytosis appear precisely at the onset of a complicating pleurisy or pneumonia in the course of typhoid fever.

The course of the leucocytosis in pneumonia is extremely interesting. Increasing with the lung-consolidation, it reaches its maximum just before the crisis, and then the decrease in the number of leucocytes is as marked as the fall in temperature. Thus, a leucocytosis of 20,000 to 30,000 may drop within a few hours to 6000 or 8000. There is some reason for believing that the greater the degree of local reaction (of which the leucocytosis may be regarded as an index) in a disease like acute lobar pneumonia, the less is the virulence of the general blood-poisoning. Thus Tschistovitch claims that in a pneumonia where the leucocytosis is slight or absent the termination is always fatal. The theory has received some support from von Jaksch; and if these results are confirmed, the blood-examination in pneumonia will be of great use for the prognosis. In one of my own cases, however, there was a leucocytosis of over 45,000 to the cubic millimetre just before death, the autopsy showing a croupous pneumonia of the right upper lobe, together with a fibrino-purulent pericarditis, with myriads of the lanceolate pneumococci in the exudate. In a recent fatal case there was a leucocytosis of 114,000 to the cubic millimetre. The disappearance of the leucocytosis in erysipelas is, as in pneumonia, also by crisis.

In addition to these inflammatory leucocytoses, a large, sometimes enormous, increase of the leucocytes has been observed in the cachexias of malignant neoplasms. How far this leucocytosis is dependent on the local reaction in the neighborhood of the tumor (necrosis and wandering-in of leucocytes) is not as yet clear.

LEUKÆMIA.

Definition.—A peculiar disease, assuming various forms, characterized by a persistent increase in the number of the white blood-corpuscles, associated with alterations occurring either singly or together in the spleen, lymph-glands, and bone-marrow.

History of the Affection and its Different Forms.—Though the claim to priority has been much disputed, it is now generally agreed that Virchow was the first to recognize the increase in the white blood-corpuscles as an essential feature of the disease. He gave it the name leukæmia. Virchow's article appeared in November, 1845; in October of the same year Bennett and Craigie had described a case in the *Edinburgh Medical Journal*, but thought that the peculiar blood-condition was due to the presence of pus, and accordingly spoke of it as a suppuration of the blood. Other cases had been observed by Piorry and Rokitansky, who also believed that they were dealing with a pyæmic condition. Vogel in 1849 was the first to diagnose the disease during life. In 1851, Bennett collected additional cases, and gave the name of leucocythæmia to the disease. In the following year the same authority published a monograph on the subject, claiming for himself priority in the discovery of the affection—a paper which aroused a lively controversy between the professors of Edinburgh and Berlin.

Virchow described two forms of the disease. In his *Cellular Pathology* he states that he was able to recognize one class of cases as characterized by the presence of a large number of the smaller forms of leucocytes in the blood, together with marked involvement of the lymph-glands; in another series of cases, where the spleen was much enlarged, it was the larger white blood-cell forms that were predominant. Many years later Neumann described a myelogenous form in which the bone-marrow showed marked changes. Although it has been customary to speak of lymphatic, splenic, and myelogenous leukæmias, it is rare to find a pure form of any one of these, more particularly of the two latter. So far, only one case of pure myelogenous leukæmia has been recorded (Leube and Fleischer), and whether a splenic leukæmia can occur independently of bone-marrow changes would seem doubtful. The lymphatic form is, however, sharply separated from the others both clinically and anatomically, and there would seem to be a tendency at present to group all leukæmias under two headings—(1) splenic-myelogenous and (2) lymphatic leukæmia.

Behier reported an intestinal type characterized by changes in the lymphatic apparatus of the bowel, and Kaposi in 1885 recorded a case in which the lymphatic elements of the skin were first involved—an affection to which he gave the name lymphoderma perniciosa. The cases in which the tonsillar and pharyngeal lymph-elements seem to be primarily affected are of peculiar interest, as we shall point out when we speak of the etiology of the disease.

A distinction between acute and chronic leukæmia is justifiable, but the former is much less common than the latter.

Etiology.—Notwithstanding most careful clinical study and thorough histological and bacteriological investigation, the secret of the causative factor in this disease is almost as profound now as it was half a century ago. The idea that leukæmia is a specific infectious disease (Klebs, Osterwold, Roux) has, however, gained ground during the past decade. Histologically, there are many points of resemblance between leukæmia and the infectious granulomata; and this, taken with the fact that the acute cases running a fatal course in a few days or weeks with high temperature correspond throughout to the clinical picture seen in the acute infectious processes, goes far to make a micro-organismal origin probable.

An interesting case has been recorded recently by Obrastzow, in which the attendant of a patient suffering from acute leukæmia developed the same disease and died: this suggests not only infection, but also the possibility of direct contagion, but inoculations with leukæmic blood have so far been without result.

Since Hinterberger in Nothnagel's clinic pointed out that by far the larger number of the acute leukæmias are accompanied by a stomatitis or by intestinal ulceration, we are tempted to regard these not as complications, but rather as primary affections affording a gateway of entrance (infection-atrium) for the leukæmic virus. A patient with splenic-myelogenous leukæmia, who entered my clinic in August, 1892, gave a history of a dysenteric attack two years previously—*i. e.* in the summer of 1890—but dated his splenic enlargement only from February, 1892: while the splenic tumor must have existed longer than this, yet the cases in which a leukæmia has been preceded by intestinal lesions occur far too frequently to allow us to regard the circumstances as merely accidental. Troje has speculated further, and suggested that in the chronic leukæmias, where enlargement of the cervical lymph-glands occurs early, the virus may have entered by way of the swollen tonsils. At a recent medical meeting in Berlin,[1] Troje advanced the theory that the so-called pseudo-leukæmia represents a leukæmic condition, the forerunner of a true leukæmia. While cases which have been definitely diagnosed as pseudo-leukæmia have, under the eyes of thoroughly competent clinicians, been observed to pass on into true leukæmia, yet the statistics are too limited to permit us to hold that such a relation is constant. The peculiar disease occurring in children under two years of age first described by von Jaksch (who named it "anæmia infantilis pseudo-leukæmica"), and subsequently by Luzet, in which clinically one finds the spleen somewhat enlarged, the blood oligocythæmic with an increased number of white blood-corpuscles, would seem to stand in an intermediate position between the pseudo-leukæmia of Hodgkin and Trousseau and the true splenic-myelogenous leukæmia: where the affection is not terminated early by an associated gastro-enteritis, the blood may assume the characteristics of a true leukæmia, the so-called "hypertrophic" leucocytes being present.

So far, no micro-organism has been definitely proven to be the cause of the disease: the monads of Klebs, the cocci-like bodies described by Byrom Bramwell, the bacilli of Majocchi and Bicchini have probably nothing to do with the affection. Kelsch and Vaillard report a case in which they found bacilli, and recently Pawlowsky of Kiew[2] describes a bacillus which he claims is peculiar to leukæmia and which he found in six successive cases. The organism grows in short rods with rounded ends, and can be cultivated, although not on the ordinary media. He has not been able, however, to reproduce the disease by injection into animals.

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[1] *Deut. med. Woch.*, 1892, No. 16.

[2] *Deut. med. Woch.*, 1892, No. 28.

What the true nature of the disease is, is a question which must still be left open ; scarcely any one now-a-days looks upon it as a primary blood-disease ; on the contrary, the blood-condition is generally regarded simply as an expression of the pathological changes going on in the hæmopoietic organs.

Leukæmia is not uncommon on this continent. Of the 18 cases of which I have notes, 11 occurred in Montreal, 2 in Philadelphia, and 5 within the last two years at the Johns Hopkins Hospital. It does not seem to be more frequent in the South.

It may occur at any age, though most common in the middle period of life. I have seen a case at eight months, and it has been known to occur as early as the eighth or tenth week and as late as the seventieth year. Eleven of my cases were males, and the disease is undoubtedly less frequent in females ; thus of 200 cases collected by Birch-Hirschfeld, 135 were males and 65 females.

Although the majority of the patients come from the lower and middle classes, no definite influence can be attributed to social and sanitary conditions. Mental worry and depression have been mentioned as predisposing causes. A history of injury—a fact to which De Chapelle has drawn especial attention—is not infrequent : 3 of my patients laid great stress on this, 2 ascribing their disease to having strained themselves by over-lifting, and 1 to the effects of a kick from a horse. It seems quite improbable that traumatism by itself could set up a process of this kind.

That malaria and syphilis bear any etiological relation to leukæmia is scarcely probable, although in 150 cases analyzed by Gowers, 30 had a malarial history, and over one-third of my cases had previously suffered from malarial invasion.

In female patients the affection most often develops at the climacteric, though pregnancy would seem somewhat to predispose to it. A patient of Cameron's at Montreal passed through three pregnancies, bearing on each occasion a non-leukæmic child. That heredity, however, plays some part is shown by this same case, since the grandmother, mother, and one brother of the patient suffered from symptoms strongly suggestive of leukæmia, and two of her children died of the disease. A leukæmic child may, on the other hand, be born of a healthy mother (Sanger).

Dogs not infrequently have leukæmia, and it has been described in horses, oxen, cats, swine, and mice (Bollinger, Eberth).

Symptomatology.—The onset (in the splenic myelogenous leukæmia) may be so insidious that the splenic tumor may fill a large part of the abdomen before the patient suffers much inconvenience. As a rule, it is for this "lump in the side" that he first consults the physician, or perhaps he notices that he has been getting short of breath and has palpitation of the heart, pallor, and other symptoms common in anæmia. Epistaxis and gastro-intestinal symptoms often occur early ; they may even precede the onset of the disease.

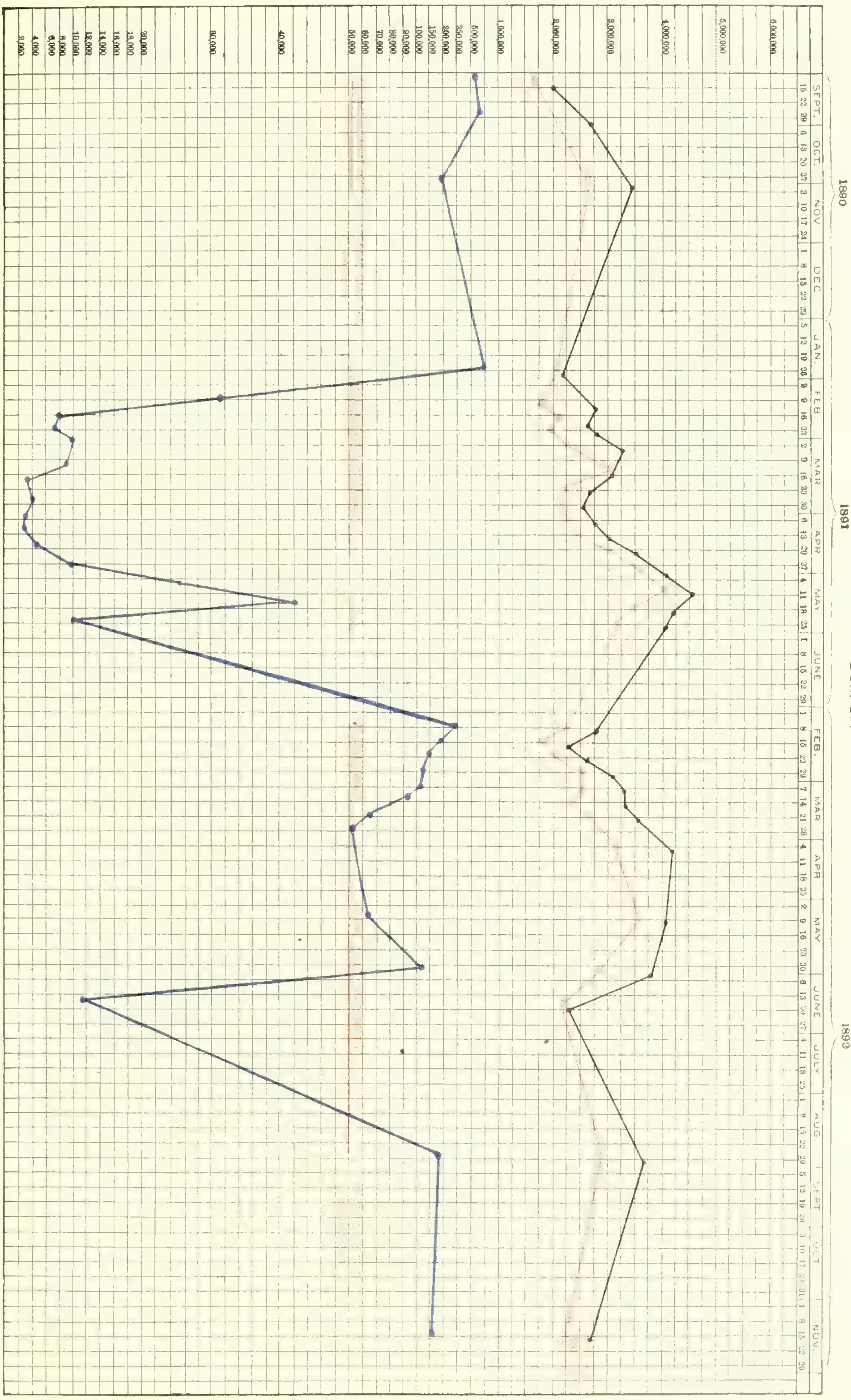
On the occurrence of a previous dysentery we have already remarked. There are instances in which a sudden or fatal hæmorrhage has been the first symptom. In one of the cases of my series a boy who died of hæmatemesis had been two days before apparently quite well, and had played in a game of lacrosse.

The symptoms referable to the stomach, such as a feeling of oppression after eating, nausea, and vomiting, are rarely absent. The vomiting, in fact, often appears early, and is at times a troublesome feature. The bowels are usually loose, diarrhœa frequently occurring early in the disease: this is said to be more frequent in those cases in which the lymph-follicles of the intestines are involved. The stools are thin and watery; in some cases there is a true dysenteric process in the colon, with tenesmus, and mucus and blood are seen in the fæces.

The liver becomes enlarged at some stages of the disease; jaundice is not common, but may result from obstruction due to catarrhal inflammation of the duct or to pressure of the glands in the hilus of the liver. Ascites may be a prominent symptom, and is probably due either to the splenic tumor or to the pressure of enlarged glands on the portal vein. Willcocks has described a leukæmic peritonitis due to new growths in the membrane.

The Blood.—No matter what the form of the disease, it is the blood examination alone that offers distinctive features. We have already mentioned the different forms of leucocytes, and the relative proportion of those of each form to the whole number in health. In the lieno-myelogenic forms of leukæmia the most striking blood-change is the enormous increase in the number of the white cells. Instead of the normal proportion of 1 white to 500 or 1000 red cells, the proportion in leukæmia may be 1 to 10 or 1 to 5, or the two kinds may occur in equal numbers; indeed, there are cases recorded in which there were actually more colorless than colored elements. A drop of blood from the finger-tip in a well-marked case will be more or less turbid, reddish-brown in color, or in extreme cases possibly chocolate-colored. Very often a single glance through the microscope at the fresh blood-slide will settle the diagnosis. Much discussion had taken place before we were well acquainted with the affection as to how great an increase there must be in the number of white blood-cells, and what relation of whites to reds is necessary to constitute a leukæmia. After this, when Ehrlich had pointed out that as a rule the eosinophilous cells were increased in the disease, clinicians went astray in regarding an increase in those cells as pathogenic of leukæmia. While it is true that they are generally both relatively and absolutely increased, this is by no means always the case, and besides, we must not forget that their number may be as great or even greater in certain other affections, and even at times in health. The lymphocytes (Plate I, Fig. 1, *b*, *b*, *b*) are relatively diminished in number; instead of making up 20 or 30 per cent. of the whole number of white cells, these small mononuclear forms may be reduced to less than 1 per cent. The leucocytes with polymorphous nuclei and neutrophilic granules (Plate I, Fig. 1, *e*, *e*) may be present in normal proportions; usually, how-

FIG. 11.



Blood-Chart of Case of Spleno-myelogenous Leukæmia. Black, red corpuscles; Red, hæmoglobin; Blue, colorless corpuscles.

ever, they are relatively diminished, and may, especially in the later stages, be very few in number. In the dry preparations the numerous brightly-stained eosinophiles (Plate I, Fig. 1, *f*) form a striking picture, but in this variety of leukæmia the most important characteristic of the blood is the presence of certain cells which may be said not to occur at all in normal blood. These cells closely resemble the large mononuclear leucocytes, but differ from them in several important particulars. Ehrlich has studied them with great care, and first described them as large mononuclear forms containing a fine thickly-set ε-or neutrophilic granulation. Believing that they originated in the bone-marrow, he has named them myelocytes. (Plate I, Fig. 2, *g*.) In one of my cases, in which the blood was examined by Thayer according to Ehrlich's method, these myelocytes at one time made up nearly 25 per cent. of the whole number of white corpuscles. H. F. Müller has described large white elements in the blood in this disease, varying in size, but being usually one-third to one-half larger than the ordinary white cells; the nucleus is large, plump, and oval, and usually lies excentrically, and its intranuclear network is more delicate than that of the ordinary leucocytes; the cell-body is often surrounded by a slightly thickened layer of cell-substance. Mitoses were numerous in these cells, and Müller has shown that precisely similar cells and karyokinetic figures occur in the bone-marrow. Similar cells had also been described earlier by Cornil and were named by him *cellules medullaires.*

My own study has convinced me of the importance of this peculiar cell-form for the diagnosis of a myelogenic leukæmia, and I have no doubt that Ehrlich, Cornil, and Müller have been describing under different names the same morphological element.

Occasionally there are leucocytes in the blood of leukæmic patients which contain basophilic granules; they are by no means always to be found. As a rule, there is no marked oligocythæmia, the red corpuscles rarely going lower than two millions per cubic millimetre; the amount of hæmoglobin corresponds to the decrease or is reduced in a somewhat greater proportion. Nucleated red blood-corpuscles are present, and, as a rule, in considerable numbers; they are chiefly normoblastic in type (Plate I, Fig. 2, *h*), but megaloblasts (Plate I, Fig. 2, *i*) occasionally occur. Cases with the blood of the type of a pernicious anæmia have subsequently developed a true leukæmia.

In the pure lymphatic leukæmia the blood-condition is quite different. Here one never meets with the enormous increase in the number of white cells described as characteristic of the ordinary form, a proportion of 1 to 10 being rarely exceeded. The increase takes place solely in the small mononuclear elements (lymphocytes), the large mononuclear and polynuclear forms being relatively greatly diminished in number. The enormous disproportion is well shown in a case described by Uthemann, where 93 per cent. of all the white cells were lymphocytes. Eosinophilous cells and nucleated red corpuscles are rare; myelocytes are, as a rule, not present unless there be an associated disease of the bone-marrow.

Just as there are mixed forms of leukæmia, however, so the blood-condition may deviate from the two types above given. In a case of acute leukæmia which came into my clinic at the Johns Hopkins Hospital, where the glands as well as the spleen and bone-marrow were affected, in the blood, besides a large proportion of lymphocytes and myelocytes, many large mononuclear leucocytes were present.

In leukæmic blood one not infrequently finds a number of polynuclear leucocytes devoid of the usual ε-granules—a phenomenon as yet not understood. The want of amœboid movement in the white cells of the blood in leukæmia was first pointed out by Cafafy; this is particularly noticeable when one compares leukæmic blood with normal specimens on the warm stage. The explanation is easy to find, since we know that of the leucocytes in health it is only the polynuclear forms which have active amœboid movements. Other points to be mentioned are—(1) the abundance of blood-plates in many cases; (2) the unusually dense fibrin network between the corpuscles; and (3) the appearance of Charcot's octahedral crystals in blood-slides which have been kept for a short time.

The Circulatory System.—It is rare that there are symptoms referable to the heart, although the apex-beat may be displaced considerably upward by the enlarged spleen. The pulse, though large in volume, is usually frequent, soft, and of low tension. Œdema of the ankles, or even general anasarca, may occur toward the end of the disease. Hæmorrhages are common in all stages, those from the nasal mucous membrane being the most frequent.

In two cases which I have seen fatal hæmatemesis occurred before there was any suspicion of an existing leukæmia. Hæmoptysis and hæmaturia are rare; cerebral apoplexy was the cause of death in one case of my series. The "leukæmic retinitis" is another manifestation of the hæmorrhagic tendency.

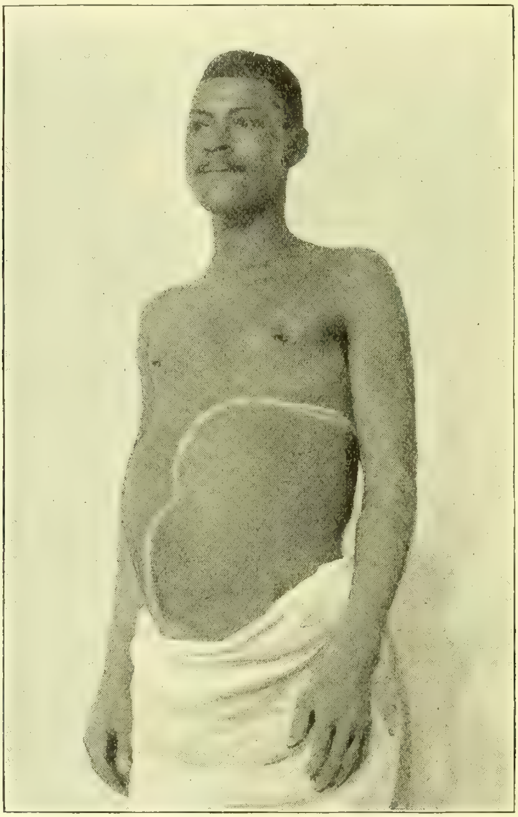
Other Organs.—The shortness of breath is due, as a rule, to deficient oxygenation owing to the oligocythæmia. The lungs are scarcely ever responsible for any symptoms until toward the end of the disease, when œdema or pneumonia may carry off the patient.

If we except the cerebral symptoms, such as headache, dizziness, and fainting spells, which are associated with anæmia, the central nervous system does not seem to be much affected. We have already spoken of the occurrence of sudden coma following hæmorrhage from one of the cerebral vessels. The peculiar retinitis, which is by no means rare, consists chiefly of hæmorrhagic patches, but there are also sometimes true leukæmic new growths with aggregations of round-cells in the retina. A genuine optic neuritis is not common. The hearing is frequently affected, and deafness may come on early in the disease. As a rule, the temperature is more or less elevated, ranging at times as high as 102° or 103° F., but periods of pyrexia may alternate with prolonged intervals of normal temperature.

The urine may be albuminous, but it is by no means always so. There is an abnormal increase in the amount of uric acid excreted, which Salkowski believes may stand in direct relation to the splenic enlargement. The cause of the persistent priapism which often occurs is unknown. In a case recorded by Edes it was the first symptom; it may persist for days or even weeks (Peabody).

The Spleen.—When a patient suffering from lieno-medullary leukæmia first consults the physician, the spleen is nearly always much enlarged, and, though usually somewhat tender, may give rise to very little inconvenience. Its border will be felt in the left side of the abdomen as a hard, smooth, rounded lump which rolls over the finger-tips with each full inspiration. The organ when much enlarged may extend as far as the navel, and I have even seen it fill the whole left side of the abdomen and extend into the right iliac fossa. (See Fig. 12.) One can sometimes feel a friction fremitus over the tumor, and on auscultation, besides the rub, a "splenic souffle," systolic in rhythm, may sometimes be heard. Gerhardt has described a pulsating spleen in one of his cases of leukæmia.

FIG. 12.



Case of Lieno-medullary Leukæmia—showing enlargement of spleen.

That the tumor is the spleen is, as a rule, easily decided from the position, form, and feel of the enlargement; moreover, not infrequently one is able to clearly make out a notch, or even several notches, in the anterior border. It varies much in size from time to time; after meals it is increased, after hæmorrhage or severe diarrhœa diminished. As might be expected, pressure-symptoms sometimes occur, such as distress after eating or obstruction of the bowels, from the latter of which death has been known to result.

The Lymph-glands and Bones.—In ordinary leukæmic patients it is rare for the lymph-glands to be much enlarged: after the spleen has become very large the lympatic glands may increase in size, but even then it is the superficial ones which are generally affected. One never sees such huge bunches of them as are found in the pseudo-leukæmia of Hodgkin. In pure lymphatic leukæmia the lymph-glands are generally enlarged, and the spleen perhaps only slightly so, but the disease is a very rare one indeed, and needs further study.

There may be scarcely any tenderness over the bones during life, which at autopsy show the most advanced marrow-changes; the patients sometimes complain when the sternum is percussed in the course of the physical examination. Irregularity and deformity of the ribs, the sternum, and other flat bones occasionally result from the leukæmic bone-changes.

Morbid Anatomy.—The body is pale and may be much emaciated; œdema and dropsical effusions are common. When the heart or aorta is opened, the blood is usually found to be clotted, the clots having a peculiar greenish-yellow color, reminding one of the fat of a turtle. When the increase in the white elements has been extreme, the color of the clotted masses may be yellowish-white, and it has more than once happened (as in a case of Virchow's) that the observer on opening the right auricle has believed for a moment that he had before him an abscess. The large amount of blood in the heart and vessels is a noteworthy feature; in one of my cases the heart-chambers alone contained blood-clots weighing 620 grammes. All of the vessels were engorged in the same way, the portal vein just above the union of its branches measuring 11 cm. in circumference.

The blood has been examined chemically by various observers with results which do not altogether correspond. Scherer as early as 1852 found hypoxanthin, lactic acid, formic acid, acetic acid, leucin, and tyrosin present, and the diminution of the alkalescence of the blood (it is even acid at times) has been supposed to be due to organic acids. The presence of a notable quantity of peptones has been mentioned by Salkowski, and confirmed recently by Freund and Obermayer.

The octahedral crystals which are found in blood which has been allowed to stand for some time were first described by Charcot and Robin. Their nature is as yet imperfectly understood: some have thought them to be tyrosin, but Gamgee claims that they represent a phosphate of the same organic base discovered by Schreiner in semen and other animal fluids.

The specific gravity of the blood is lowered—varying from 1035 to 1050—and the watery constituents are increased. We possess as yet no satisfactory knowledge of the condition of the albuminous bodies and salts.

On examining the heart it will be found pushed up the distance of an intercostal space; the serous membranes (peri- and endocardial) not infrequently present ecchymoses, and leukæmic new growths may exist there as well as on the peritoneum. The cavities of the heart are, as a rule, dilated and the myocardium soft; if the papillary muscles be teased out, a moderate grade of fatty change is evident. Beyond an occasional fatty degeneration of the intima there is no recognizable histological alteration of the walls of the vessels.

As we have said, the spleen is nearly always enlarged. Externally there may be the signs of an old perisplenitis, with adhesions to the abdominal wall, diaphragm, or stomach, the capsule in these cases being often greatly thickened. The arteries and veins at the hilus are enlarged. The organ may vary in weight from two to eighteen and a half pounds, the latter weight being the heaviest on record. On section the spleen is firm and tough and the surface a reddish or purplish brown. There may be throughout the organ hæmorrhagic infarctions or areas of a rusty, reddish-brown color, the site of old extravasations. The Malpighian bodies are not prominent; indeed, they are, as a rule, not recognizable. On the other hand, grayish-white, well-defined lymphoid tumors may occur throughout the organ, contrasting strongly with the reddish-brown ground-substance. If the spleen be seen in an early stage and before the disease has progressed far, it will be found softer, and there will be swelling of the pulp and proliferation of the cellular elements; rupture of the spleen, it is said, has happened at this period from the excessive hyperæmia. Microscopical examination shows this proliferation to be general; karyomitotic figures and large myeloplaques are seen here as well as in the lymph-glands and bone-marrow.

The lymph-glands may be enlarged in the chronic form of leukæmia, but the hyperplasia is not extensive. In the acute lymphatic leukæmia all the lymph-glands of the body may be involved, the cervical, axillary, mesenteric, and inguinal groups most frequently, less commonly the bronchial and mediastinal. The glands are soft and easily movable, scarcely ever being matted down by a periadenitis. As in other lymph-gland affections, the size varies from time to time, often diminishing notably before death. On section the grayish color of the healthy gland is not seen; instead, the surface usually has a grayish-red color, and there are evidences of hæmorrhages into the substance.

The pathological changes in the bone-marrow have been closely studied since the investigations of Neumann. That observer claimed that the medulla of bones was affected in every case of leukæmia; there are instances, however, where no such changes are recognizable. In the majority of examples of the lieno-medullary form described by the Germans one finds that the normal fatty marrow undergoes marked alterations. Examination of, say, one of the middle lumbar vertebræ or the extremities of the long bones reveals a dark reddish-brown substance quite different from that seen in health. Occasionally there are hæmorrhagic infarctions, as in Ponfick's case, and as a result of the proliferation there may be expansions here and there of the bony case, forming localized swellings. Smear cover-glass preparations, dried in the air, heated and fixed with Flemming's solution or with picric acid, and subsequently stained with safranin or dilute hæmatoxylin, show most beautifully the cellular elements and their nuclei. Müller thus convinced himself of the identity of the medium-sized marrow-cells with the large uninuclear cells in the blood. Many mitoses were seen, and myeloplaques containing leucocytes in their interior. The theory that these latter represent an endogenous formation of leucocytes cannot be supported,[1] for we are probably here dealing with cell-inclusions. Nucleated red blood-corpuscles and eosinophilous cells are abundant. The function of the huge giant-cells of the marrow, whether in health or disease, is still unknown.

Wherever there are lymphatic elements in the body there may be leukæmic changes; thus the thymus gland, the solitary and agminated follicles in the intestine, the tonsils, the lymph-follicles of the tongue, pharynx, and mouth, may participate in the process. Even the little collections of lymph-corpuscles in the different organs, in the lungs, liver, kidneys, etc., may undergo proliferation.

The liver is frequently enlarged. Welch has described a case in which it weighed over thirteen pounds. Histologically, the enlargement is seen to be due to a diffuse leukæmic infiltration; the capillary ectasis is extreme, and the columns of liver-cells are widely separated by the crowds of white elements in the blood-vessels.

The large leukæmic tumors, though rare—being seen only in 1 case in my 12 autopsies—deserve more than a passing mention. When they are present in the organs, they appear as grayish-white nodules, and show microscopically numerous cells undergoing karyomitosis. It has been supposed that these formations arise from cells which have emigrated from the blood-vessels, but since the cell in the leukæmic new growths does not at all resemble the polynuclear leucocyte, the only one supposed to leave the vessels, this view seems improbable.

Diagnosis.—This rests entirely upon the blood-examination, and in the majority of instances is easily made. In doubtful cases the differential count of the white elements by the methods of Ehrlich should not be neglected; in hospital practice a color-analysis should always be made. Aside from the blood-condition, the clinical features may be indistinguishable from those of an ordinary splenic anæmia or Hodgkin's disease. That the disease may be present even when the leucocytes are not increased in number is well illustrated by the course of a case the chart of which is given at p. 217, and the spleen outline at p. 220. The patient, a negro, was first seen in the fall of 1890, at which time he had 2,000,000 red and 500,000 white blood-corpuscles per cubic millimetre, with 30 per cent. of hæmoglobin. He neglected treatment until January 29, 1891, when he began to take arsenic regularly in increasing doses, and in twenty-three days—*i. e.* on February 21st—the number of white cells to the cubic millimetre was found to have fallen from 714,000 to 7500, or only one-hundredth part of what they were before. The red blood-corpuscles had risen in number to 3,500,000, and the hæmoglobin had increased to 44 per cent. Could one under these circumstances, seeing the case for the first time, have made the diagnosis of leukæmia? It is here that the value of Ehrlich's methods is well demonstrated. A careful color-analysis was made by my assistant, W. S. Thayer, at different times, and the estimates given in the following table were based on differential counts of at least one thousand leucocytes at each examination:

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[1] *Vide* H. F. Müller, *Deut. Archiv. f. klin. Med.*, vol. xlvii. p. 47.

	Normal Blood.	Nov. 9, 1890.	Jan. 29, 1891.	Feb. 7, 1891.	Feb. 14, 1891.	Feb. 21, 1891.	Feb. 28, 1891.
Lymphocytes	20–30%	2.1%	0.96%	1.5%	2.7%	6.9%	10 %
Polynuclear	60–75	73.8	70	83.9	84.7	83.2	78.1
Mononuclear and Transition forms	6.0	4.6	3.0	1.5	2.1	2.5	2.3
Eosinophiles	2.4	4.8	2.3	4.2	1.6	3.0	4.4
Myelocytes	0	14.7	23.5	8.6	8.5	4.0	4.7

Now, on Feb. 21st, while the enormous enlargement of the spleen would have made one think of leukæmia, yet the mere numerical estimate or the examination of the fresh blood would have given no hint that a leukæmic process had existed. As will be seen, however, by reference to the above table, the dried and stained specimens still showed 4 per cent. of typical myelocytes, and this would have hinted at a previous existence of, and the possibility of a return of, a leukæmia.

An enormous leucocytosis might be mistaken for a leukæmic condition, but may be easily excluded by the study of stained specimens; in all ordinary leucocytoses the increase affects solely the polynuclear neutrophiles. The enlargement of the spleen in chronic malarial cachexia or malignant disease may also be differentiated from leukæmia by the blood-examination. It certainly is not justifiable to make (as has been done in at least one case of leukæmia) an exploratory abdominal incision to examine the spleen before the blood has been carefully studied.

The pure lymphatic form of leukæmia has to be distinguished from general lymphadenoma or Hodgkin's disease; in the latter, however, the glands are found in much larger bunches; and, besides, the blood-condition is in lymphatic leukæmia quite characteristic (*vide supra*).

Course and Prognosis.—In the splenic-myelogenous form the progress is slowly progressive for months and years. Recovery occasionally occurs, but this is so rare that when the diagnosis is once established we can expect death almost certainly within five years, and, as a matter of fact, the majority of cases terminate fatally in two or three years. Certain symptoms, as hæmorrhage, high fever, severe diarrhœa, and œdema, will indicate a rapid course. The progress of the affection is very irregular, and there may be transient intervals of comparative health, which encourage the patient for a time, only to be followed sooner or later by a return of the symptoms. I have known a patient (Case VIII. of my series) with an enormous spleen to get about for months, attending to a light business, when his blood-count showed a ratio of 1 white to 6 red cells.

The patient finally becomes weaker and weaker, the strength of the heart gradually fails, œdema develops, and death in most cases is the result of the general asthenia. Fatal hæmorrhage may occur at any stage of the disease. Cerebral apoplexy has been mentioned in the Symptomatology: it was the cause of death in 6 of 60 cases analysed by Gowers. Not infrequently death results from some intercurrent affection; in more than one case the autopsy has revealed an acute infection with cocci, probably an expression of the diminution of the resistance of the tissues to bacterial invasion.

The general lymphatic leukæmia appears to be more rapid in its course, and most of the acute cases of leukæmia terminating fatally in a few days or weeks belong to this class. It runs its course with hæmorrhages and pyrexia, and often resembles closely a severe scorbutus.

Treatment.—If the cases came under treatment early, we might hope for favorable results, but, as we have already said, the disease is usually well advanced before the physician is consulted. It is important to pay particular attention to the hygienic surroundings of the patient. He should have abundance of fresh air and a liberal diet, and must avoid worry and mental emotion of all kinds.

While we possess one or two remedial agents which have an influence on the disease, there is none which can be absolutely relied upon to have a good effect. The treatment by arsenic has given the best results in my hands, most cases improving, at least for a time. We must not be too hasty, however, in attributing the favorable results attained to the administration of the drug employed, since the curious remissions which often occur in the progress of the affection may thus lead to wrong conclusions. I remember one case in particular, a patient, who had been confined to bed for a long time with little hope of any improvement, without having received any special treatment became well enough after a time to get around, and even to attend to light duties. No better example of an apparently direct effect of arsenic could be advanced than the case of the colored man mentioned above. Having taken arsenic in 1891 until the relation of white to red cells became normal, he discontinued treatment and went to his home in Virginia; months later he returned, the blood-count showing marked leukæmic changes, which was again reduced under arsenical treatment. Having once decided to use arsenic, we should not be afraid to push the drug, under due precaution, till large doses are reached.

In those cases which have a decided malarial history quinine may be tried, although little is to be expected from its use. Iron and inhalations of oxygen have been recommended, and possibly may be of value in some cases. It does not seem probable that cold douches over the region of the spleen or the application of the galvanic or faradic current can do more than give the patient a certain amount of comfort and satisfaction. Still less promising is the operative treatment—the removal of the leukæmic spleen; splenectomy has been done twenty-four times for leukæmia with one recovery—namely, in the case of Franzolini. The transfusion of blood does no good, and is now scarcely ever advised. Notwithstanding the fact that the disease is almost always progressive despite the most carefully directed treatment, yet the practitioner can do a great deal to relieve the distressing symptoms. The stomach troubles and the diarrhœa should be attended to. Hæmorrhages are frequent and not rarely dangerous, and are to be checked by the usual methods. Little can be done to relieve the dragging feeling in the left side; the pain may sometimes be so severe as to call for sedatives, but their use should be delayed as long as possible. In the use of purgative medicines much caution should be observed.

HODGKIN'S DISEASE.

Definition.—An affection characterized by progressive hyperplasia of the lymphatic glands occurring with anæmia, and sometimes accompanied by the development of secondary lymphatic growths in various parts of the body.

History.—The disease which now bears his name was first described by Hodgkin of Guy's Hospital in 1832, in a paper entitled "On Some Morbid Appearances of the Absorbent Glands and Spleen."[1] Morgagni and other observers had before this mentioned cases with enlargement of the lymphatic glands terminating fatally, but the accompanying histories are too meagre to allow of any judgment as to the nature of the maladies with which they dealt. A number of the cases described by Hodgkin were undoubtedly examples of tuberculous adenitis, but at least four of them were genuine instances of what we now speak of as "Hodgkin's disease." The affection received its name from Wilks in 1865, when he reported a series of cases in which, together with anæmia, there was enlargement of the lymph-glands with growths in the spleen and other organs. Virchow described the histology of lymphosarcoma in 1845, and later Cohnheim[2] discussed the pathology of the affection, giving it the name of pseudo-leukæmia, on account of its superficial resemblance to leukæmia. The studies of these investigators attracted general attention to the subject, and the literature is voluminous. Unfortunately, many cases described have to be weeded out, as the affection has been repeatedly confounded with tuberculosis, true leukæmia, syphilis, and neoplasmata of the lymph-glands. Billroth[3] endeavored to distinguish these growths, which he named malignant lymphomata, clinically from the local non-infective lymphosarcomata, stating that in the former there was no invasion of the periglandular tissues, while in the latter the new growth did not confine itself to the glands. The description of this disease given under the name of *adenie* by the great French clinician Trousseau is so accurate that the affection is now often referred to as the "pseudo-leukæmia of Hodgkin-Trousseau." Ranvier introduced the term *lymph-adenie*, and the number of other synonymous terms used is very great. Thus, Wilks has termed it anæmia lymphatica; Wagner and R. Schulz, "desmoid carcinoma;" Mursick called it lymphatic cachexia;

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[1] *Transactions Med.-Chir. Soc.*, vol. xvii, 1832.

[2] *Virchow's Archiv*, Bd. xxxiii. p. 451.

[3] *Beiträge zur Pathologischen Histologie*, Berlin, 1857.

Bonfils, "cachexia sans leucæmie;" while Southey preferred the name "adenoid disease."

The disease is not uncommon in America.

Etiology.—At present we must allow the term pseudo-leukæmia to cover certain groups of pathological conditions which before long will be recognized as definite and distinct diseases. One has only to attempt to acquaint himself with the literature of Hodgkin's disease to appreciate the hopelessly confused condition in which the matter at present stands. There is a growing convictions, especially among the German clinicians, that in at least one class of cases, if the patients live long enough, there develops a true leukæmia; and certainly there are several instances recorded in which under direct observation such a change has taken place.

Hodgkin's disease is more common in the young, over 60 per cent. of the cases occurring in persons under forty years of age. That it is an infectious process there would now seem little reason to doubt. Klebs[1] was the earliest to point out that the changes in the tissues resemble strongly those in the infectious granulomata; and Virchow and Cohnheim both looked upon the mode of formation of metastases as an evidence rather of an infectious than of a neoplastic process. Dreschfeld in a recent article lays special stress on the probable infectious nature of the disease, and calls attention to the pyrexia and hæmorrhages which occur in acute cases. The "chronic relapsing fever" described by Ebstein, and commented upon by Pel of Amsterdam, is probably to be looked upon as an acute form of pseudo-leukæmia.

So closely do the histological changes resemble those seen in certain forms of tuberculosis that some writers have gone so far as to state their belief that pseudo-leukæmia represented a modified form of lymph-gland tuberculosis. Weishaupt studied a case in which even at the post-mortem the diagnosis in a doubtful case was settled only by the finding of tubercle bacilli in the stained sections. He studied twelve true cases of pseudo-leukæmia and found tubercle bacilli in none.

What the infectious agent is we do not know. Those cases in which pyogenic cocci have been found are to be looked upon as pseudo-leukæmias with a complicating septic infection. Flexner,[2] in working up the tissues of a case from my wards, has made out certain bodies foreign to the tissues and occurring most commonly in the larger nodules. These, he thinks, may be animal parasites which possibly stand in a causal relation to the affection. Later he secured material from another case, and succeeded in finding the same bodies in the nodules in the intestines and liver. These bodies consisted of protoplasm, and contained minute particles within them varying much in shape, although the bodies themselves did not differ much in size. The bodies were easily stained with hæmatoxylin and eosin, and were readily distinguished from the tissue-cells present by their smaller size (one-third of the size of red blood-corpuscles), and from nuclear fragments by the fact that each stained particle was surrounded by a zone of protoplasm, and the intensity of the staining was not so great as in fragments of nuclei.

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[1] *Prager Vierteljahrschrift für Heilkunde*, Bd. cxxvi. p. 113.

[2] *Johns Hopkins Hospital Reports*, 1892.

Morbid Anatomy.—The pathological changes found at the autopsy will vary according to the portions of the lymphatic apparatus affected. As a rule, the lymph-glands are soft and elastic, though in some few cases they are tough and firm. In an advanced case the glands will be seen fused together into huge bunches as large as an orange or even larger. Should the patient die in the earlier stages, this matting together of the glands is not so evident, as at the onset, when the enlargement first begins, the individual glands are isolated. As a rule, there will be found to have been more or less connective-tissue proliferation about these glandular tumors, with thickening of the fibrous capsule. The cases in which the growth perforates the capsule of the gland and invades the neighboring parts, such as the muscles or skin, have been placed, as we have said, by Billroth in a separate class. On cutting into one of these new growths the surface will be found smooth and the substance of variable consistence; sometimes it is soft and juicy, while in other cases it may be firm and dry. The tumor is usually grayish-white in color, and those cases in which caseation has been reported were probably not cases of pseudo-leukæmia at all, but rather a tuberculosis of the lymph-glands. Pyogenic processes sometimes occur, especially if the growths invade the skin, the suppuration here being of course due to a complicating infection with cocci. Pus-formations in the deeper sets of glands are rare.

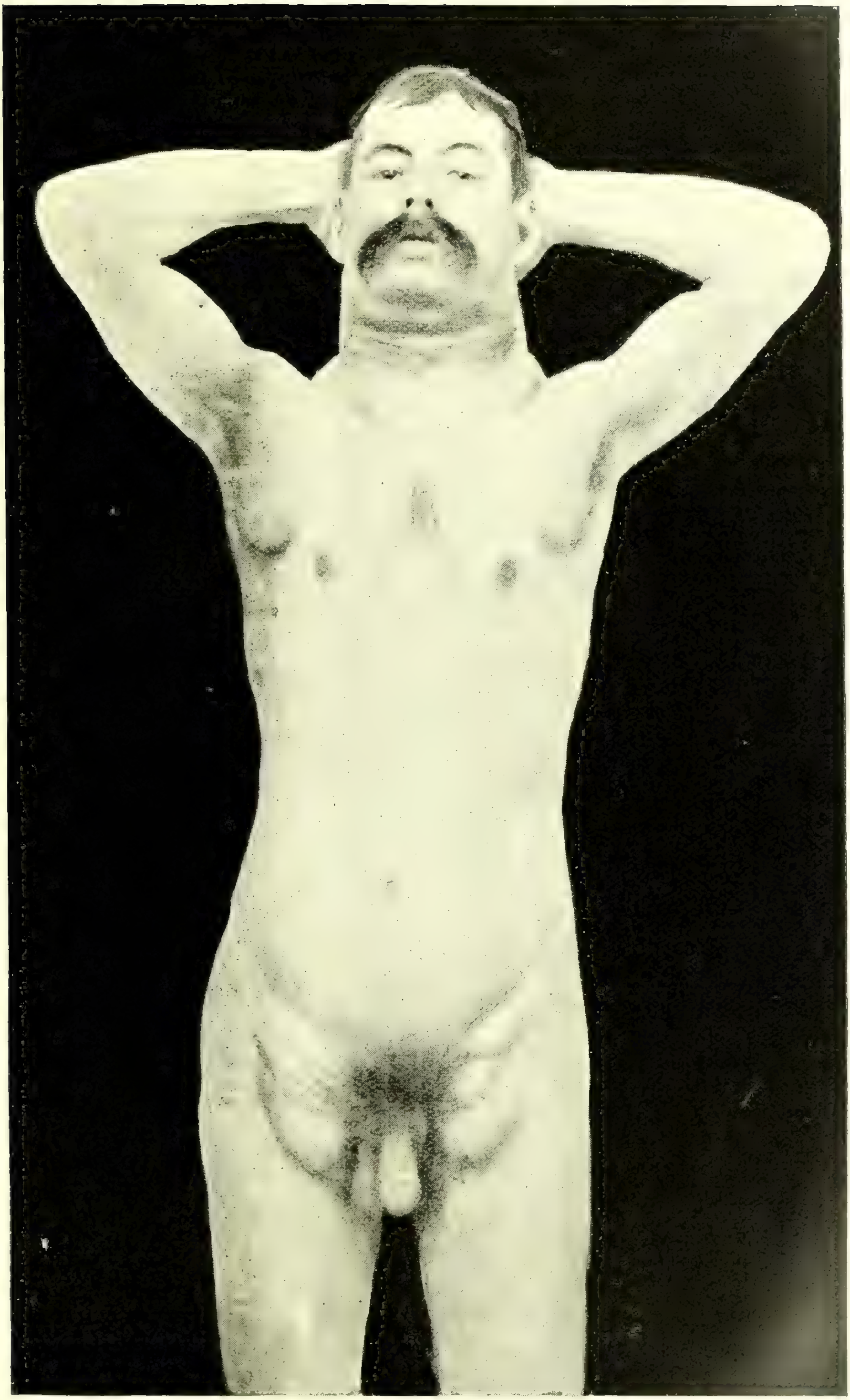
The glands of the body most frequently affected in this disease are the superficial chains, particularly those of the neck. Not infrequently, at the post-mortem examination one is able to trace the cervical glands as continuous chains running down along the trachea and large vessels to join the axillary and mediastinal glands. Next to the cervical groups the axillary glands are most frequently attacked, and then the tumor-masses may extend in under the pectoralis major and minor muscles and backward beneath the scapula. Less often such masses are formed from the glands in the groin. Those cases are particularly interesting in which the glands in the thorax are much enlarged and press on the vessels, or even occasionally perforate the sternum and appear externally as a tumor-like projection.

The retroperitoneal and mesenteric—in short, any of the lymph-glands of the body—may be involved. The diagnosis when the abdominal glands alone are implicated is of course extremely difficult. I remember when in Germany some years ago seeing a leading gynæcologist perform a laparotomy for an abdominal tumor, presumably a myoma of the uterus: the incision revealed masses of enlarged lymph-glands adherent to the uterus and adnexa, the case proving to be one of Hodgkin's disease.

The histological changes in the glands seem to consist chiefly of a hyperplastic proliferation of the cells, the reticulum, as a rule, not being thickened. The normal relation of the lymph-paths are in the early stages maintained, and it is only when the growths have become large that these are disturbed. The bands of reticular tissue vary in thickness and density in different places.

PLATE II.

(Fig. 13.)



Case of Hodgkin's Disease, showing Enlargement of Superficial Lymphatic Glands.

Besides the changes in the lymph-glands proper, the lymphatic tissues throughout the body may be affected. First among these come the follicles in the spleen: in 75 per cent. of the cases tabulated by Gowers there was some enlargement of this organ, and in 56 per cent. there were new lymphoid growths, grayish-white bodies varying in size, and consisting wholly of lymph-corpuscles supported by a delicate reticulum. The bone-marrow, the tonsils, the lymphoid follicles at the root of the tongue, the patches of Peyer, and the solitary follicles in the intestines, all may participate singly or together in the process. In an obscure case which came into my clinic in August, 1891, with abdominal pain, vomiting, and slight tympanites, the patient dying suddenly in collapse, the autopsy revealed three lympho-sarcomatous nodules in the small intestines, with similar new growths in the liver and kidneys. In this case, too, there was a diffuse atrophy of the mucous membrane of the stomach and small intestines.

The lungs are occasionally involved, either by direct ingrowth from the glands at the root or by secondary nodules similar to those seen in the spleen, liver, and kidneys. The skin is sometimes the seat of lymphomatous growths, and cases in which this occurred have been reported by Greenfield and Arning; the latter excised one of the tumors and made the diagnosis by microscopical examination. A case in which the heart-muscle contained a nodule has been described by Wiegandt. The central nervous system, the genitals, and adrenals are rarely invaded.

The distribution of the metastases necessitates the assumption of the conveyance of the exciting agent through the blood-current.

Symptomatology.—Since at present we are forced to include under the name of pseudo-leukæmia conditions varying so widely in a pathological sense, it will of course be impossible to lay down a typical and definite series of symptoms applicable to all cases. We shall therefore describe first the appearances presented in those cases in which there is an anæmia associated with enlargement of the superficial and deep glands, and which therefore correspond most closely to the affection described by Hodgkin.

The patient's attention may be first drawn to the granular tumors (see Fig. 13), or, less frequently, it is the anæmia and other constitutional symptoms which lead him to consult the physician. The cervical glands are generally the first attacked, and often on one side of the neck alone. When seen early it may be impossible immediately to exclude tuberculosis or syphilitic adenitis. I have seen a case in which the glands on one side of the neck were involved for three years before any other similar tumors appeared. Gowers speaks of a boy in whom Heath excised the glands from the axilla, which had been enlarged for six years; he was seen four years later, and by that time the disease had spread only as far as the cervical glands on the same side. When the deeper glands are first affected, symptoms resulting from the pressure of the new growths on the vessels or nerves may be the earliest evidences of disturbances; thus, enlargement of the bronchial and tracheal glands may cause marked dyspnœa and thoracic pain before anything definite can be made out by physical examination. In a case observed by Ross of Montreal, (on which I made an autopsy) in which œdema of the feet and lancinating pains in the distribution of the nerves, occurring early, were followed by complete paraplegia, we found a gland-mass pressing on the spinal cord. If the axillary glands be much enlarged, there will be œdema of the hand and arm from venous obstruction. The inguinal glands sometimes form large tumors which occasionally become pedunculated.

Extraordinary symptoms from the pressure of intrathoracic glands are at times met with. In a man who still frequents my clinic the superior vena cava is completely obliterated. There is considerable congestion of the head and upper extremities, but a fairly good compensatory circulation has been established through the superficial veins. The chest-walls have been converted into a huge felt-like mass of dilated veins, the latter emptying into two large trunks, the dilated epigastric veins. Occasionally he has an attack of phlebitis in one of the smaller veins, and here and there small phleboliths have been formed. One day, while doing heavy lifting, he had an attack of hæmoptysis, losing about a quart of blood—an accident which relieved the congestive symptoms for some little time.

The retroperitoneal glands are more frequently enlarged than the mesenteric, and in thin individuals the nodules in the abdomen can be made out. Along with the affection of the abdominal glands there may be well-marked bronzing of the skin, as in Case IV. of my series; Féréol described a similar case, and Crocq suggests, by way of explanation, pressure of enlarged glands on the suprarenal capsules.

The variation in the rate of growth and in the size of the glands at different times is astonishing. Where they have been large they may diminish in volume or even entirely disappear; a rapid diminution in the size of the glands shortly before death has been frequently observed.

The spleen is often sufficiently enlarged to be easily palpable in the left hypochondrium. The thryroid is occasionally affected, and in rare instances the thymus as well.

The patient may go on for a long time complaining of little else than the inconvenience resulting from the presence of tumors. Sooner or later, though, the anæmia and the cachexia gradually appear. He begins to feel languid and disinclined for exertion, whether mental or physical; œdema of the legs, headache, palpitation, and dyspnœa succeed. The blood-count is, as a rule, not strikingly low; in only one instance have I seen the red blood-corpuscles sink lower than 2,000,000 per cubic millimetre and often there is no decrease at all in the number of red blood-corpuscles. There is no extreme poikilocytosis and the leucocytosis is inconsiderable. In the differential count the lymphocytes appear to be relatively increased. Where there is a marked leucocytosis with a preponderance of lymphocytes, we have to think, of course, of a lymphatic leukæmia. An occasional nucleated red cell (normoblast) may be found.

The palpitation of the heart may or may not be severe. On examination murmurs may usually be heard over the cardiac area, but these are probably only functional in character. The dyspnœa may be the result of the anæmia or be due to pressure on the trachea; occasionally there is hydrothorax or œdema of the lungs. If the temperature be carefully recorded, more or less fever will be found even in the earliest stages. It may be continuous, but it much more often has a recurrent type. In Case I. of my series there were remarkable ague-like paroxysms at varying intervals. These attacks, which may persist for weeks or even months, have been studied also by Ebstein and by Pel, the former observer believing in one case that he had discovered a new infectious disease, since during a period of nine months the patient had attacks of fever lasting from ten to fourteen days, alternating with apyrexia for ten or eleven days.

Usually the digestive symptoms are not marked, although in those cases in which there is extensive atrophy of the gastro-intestinal mucosa one would expect to find serious disturbances. There may be a little ascites, and the liver is slightly enlarged. Deafness may be consequent on adenoid growths in the naso-pharynx which have occluded the orifices of the Eustachian tubes. Unilateral disturbances from pressure on one cervical sympathetic, showing itself by dilatation of one pupil and flushing and sweating of one cheek, may be seen. In addition to an actual invasion of the skin by lymphomatous growths and the bronzing which we have already mentioned, there is occasionally an intense and troublesome pruritus with or without a papular rash. Sometimes there is albuminuria, and in the acute cases there may be hæmorrhages into the skin and mucous membranes. We have already spoken in our remarks on Leukæmia of the peculiar pseudo-leukæmic anæmia which has been described by von Jaksch and Luzet as occurring in children.

Diagnosis.—We have to carefully exclude syphilis. Even when no history of chancre can be obtained, the patient must be questioned with regard to ulceration of the throat, falling out of the hair, and if it be a woman one should ascertain whether or not she has had miscarriages. The differentiation from tuberculous adenitis may be even more difficult. The chief points of distinction are as follows: Tuberculous adenitis is more common in the young, and involves the submaxillary group of glands oftener than those running along the anterior and posterior border of the sterno-mastoid, whereas these latter are more frequently the first attacked in Hodgkin's disease.

A long-standing affection of one group of glands without an extension of the process to others is suggestive of tuberculosis rather than of lymphadenoma. But the most important point of all is. that the tuberculous glands tend to suppurate—a feature rarely seen in pseudo-leukæmic glands unless they have reached an enormous size. We must not forget that there is such a thing as an acute tuberculous adenitis involving the lymph-glands of the neck. As an instance of such a possible confusion we quote the following case: A man of twenty-four was admitted to the Montreal General Hospital with marked swelling of the cervical glands on both sides, tonsillitis, sloughing pharyngitis with irregular fever and diarrhœa: a diagnosis of Hodgkin's disease was at first made, but was afterward given up. The occurrence of ague-like paroxysms and of recurrent attacks of fever is in favor of pseudo-leukæmia, but there certainly are cases in which it may be impossible for a time to make a certain diagnosis. Where the glandular enlargement is localized, it is not only justifiable, but also advisable, to remove them, when the diagnosis can be cleared up by microscopical examination.

The disease is to be distinguished from genuine leukæmia by the examination of the blood, which should be made carefully in all cases.

Prognosis.—Hodgkin's disease is in the long run almost invariably a fatal affection. There may be marked variations in the course, distinct exacerbations and remissions being characteristic, but the cases of complete and permanent cure are rare. Where the gland-involvement remains localized for a long time and does not extend to other groups, we may expect a long period of comparative health, but when the gland-tumors are multiple and are to be found in different regions of the body, and especially in those cases in which the cachexia develops early, a fatal termination may be soon expected. Death comes in different ways, but most frequently it is the result of the general asthenia: the patient grows weaker, and perhaps becomes emaciated; the dyspnœa and palpitation increase, the legs become swollen, and at the end perhaps there may be hydrothorax or œdema of the lungs, with heart failure. Of course death may occur from pressure upon important parts, as in those cases where the tracheo-bronchial groups of glands are much enlarged. The occurrence of high fever or of hæmorrhage is of grave import. The acute cases may die in a few days or weeks, the chronic lasting sometimes many years. Two of my cases died from general infection with the streptococcus pyogenes.

Treatment.—Besides hygienic measures, the internal administration of arsenic is certainly of value in some instances. As soon as the diagnosis has been established, the patient should be given Fowler's solution after meals, well diluted, the dose being gradually increased. If benefit is to be derived, the arsenic must be pushed until its physiological effects are noticeable; if these be troublesome, it may be necessary to discontinue the medicine for a time, returning to it later when they have passed off. The injection of arsenic into the affected glands by means of a hypodermic needle has been recommended, but we should be inclined to regard it as a wholly unnecessary procedure.

When the case is seen early and only a few glands are enlarged, without any evidence of increase in the size of the spleen, the question of excision of the nodules may arise, and, if there be also no cachexia, I consider it good practice. Not only does the diagnosis become entirely cleared up, but there may perhaps be a chance of limiting the progress of the disease in this way, and at any rate, with the improvements in technique possessed by modern surgeons, the operation is trifling and attended with very little danger. Surgical treatment may be required where asphyxia is threatened from pressure on the trachea.

The external application of substances like the tincture of iodine or the ointment of the biniodide of mercury can have no permanent effect. The internal administration of iodine and of the iodide of potassium would not seem to be of any greater utility. Gowers and Broadbent think that they have seen good results following the use of phosphorus, and where for any reason the arsenic is not well borne this drug may be given a trial. Von Jaksch recommends inunctions of green soap and strong galvanism.

Everything possible should be done to support the strength of the patient: fresh air, nutritious food, an environment quiet and cheerful, will, with the aid of tonics such as quinine and iron, do much to add to the comfort and welfare to the individual affected. Change of air and scene, a course in a well-conducted establishment where hydrotherapy may be employed, by improving the general health may do something in holding the disease in check, but where the patient's means are limited he should not be encouraged to sacrifice too much on measures which at best can give but transient benefit.

DISEASES OF THE SUPRARENAL CAPSULES AND DUCTLESS GLANDS.

BY WILLIAM OSLER.

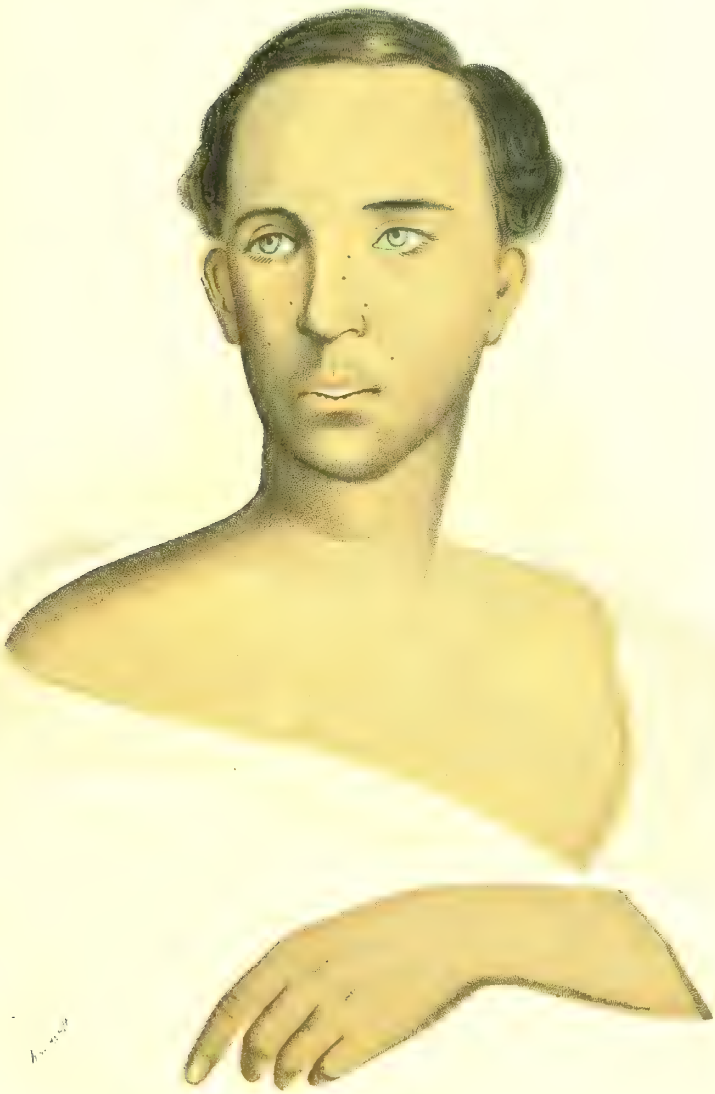
ADDISON'S DISEASE.

This disease, first recognized by Thomas Addison of Guy's Hospital, London, and described by him in 1855 in a monograph entitled *The Constitutional and Local Effects of Disease of the Suprarenal Capsules*, is associated in a majority of the cases with tuberculosis or other affections of the adrenals, usually also with changes in the abdominal sympathetic nerves. It is a constitutional affection, characterized by asthenia, a depressed state of the circulation, irritability of the stomach, and pigmentation of the skin and mucous membranes.

Etiology.—About 60 per cent. of the cases are in males. The disease may occur at almost any age, but is by far most commonly seen between the ages of fifteen and forty. There is one remarkable case recorded by Belaiëff in which the affection was congenital. The child's skin had a yellowish-gray tint, and at the autopsy, eight weeks after birth, the suprarenal capsules were found to be large and cystic. A history of traumatism, as injury to the abdomen or back, is not uncommon (Greenhow). The disease is very rare, occurring only about once in every two thousand patients at the larger clinics. In America it seems to be even less frequent than in Europe. Eight cases have come under my personal observation.

Morbid Anatomy and Pathology.—Emaciation is sometimes but not always present, the fatty tissues being generally well preserved. The suprarenal capsules are diseased in 88 per cent. of the cases (Lewin). The most frequent change is tuberculosis. The glands are enlarged, caseation being seen in some portions, while in others the gland-substance has been replaced by fibrous tissue. Sometimes the tuberculosis seems to be primary, and in very rare cases no tuberculous lesions are found in other parts of the body. On the other hand, there is much more often an associated pulmonary or bone tuberculosis, or a general miliary tuberculosis may have existed. Diseases of the suprarenals, non-tuberculous in nature—*e. g.* atrophy, carcinoma, or sarcoma—have been accompanied by the clinical phenomena of Addison's disease; and, again, there are cases in which these symptoms have been well marked during life, in which after death no lesions could be found in the adrenals. Further, in patients who during life have shown none of the symptoms of the

PLATE III.



Addison's Disease.

disease, at the autopsy advanced changes in the suprarenals have been noted. Gilman Thompson has collected 113 such cases.

It is difficult—nay, almost impossible—to harmonize facts so contradictory, and no theory has as yet been proposed which is entirely satisfactory. It has been urged that the clinical symptoms are due not to the disease in the adrenal glands themselves, but rather to the involvement of the neighboring branches of the sympathetic and its ganglia (Greenhow). Of 87 cases in which the sympathetic system has been examined probably 60 presented decided lesions (Thompson). Functional disorder in this view must be assigned in the cases in which the ganglia and nerves were found normal. Carcinoma of the adrenals less often than tuberculosis gives rise to the symptoms of Addison's disease, and it has been maintained that the former less frequently interferes with the functions of the sympathetic.

Another view is that the clinical symptoms are an expression of a disturbance of function of the adrenals *per se*. According to this hypothesis, the suprarenals in health are to be looked upon as glands whose " internal secretion " is essential to the life of the individual, either on account of the influence which it exerts on the nervous system or because it neutralizes or destroys certain poisons which are constantly being produced in the body. Such poisons, it is held, if left to themselves, give rise to those peculiar symptoms which are characteristic of the disease under discussion. Some experimental work has been done on the subject, and the recent articles by Tizzoni[1] and Alexander[2] are worthy of consideration ; but any safe conclusions regarding the relation and functions of the adrenals to the welfare of the organism as a whole cannot yet be made, and whether these bodies are important chiefly from a secretory or from a nervous standpoint must be left to further investigators to decide. As to changes in the other organs, there is not much to be said. The spleen has been found enlarged, and at times the thymus gland has been found to have persisted and to have increased in size. The heart is often small, and scarcely ever shows any hypertrophy of its wall. The pigmentary changes in the skin occur chiefly in those regions which are normally darker than others, and the pigment deposition would appear to take place in the ordinary way.

Symptomatology.—The pigmentation may attract attention before the other symptoms, although, as a rule, it is not marked until the disease is well advanced. The color varies, being of all shades from a light yellow to black ; often there is a decided bronze tint in the *melasma suprarenale*. The parts which are exposed, or those in which more or less pigmentation normally occurs, are always most deeply stained. Ordinarily, the pigmentation is diffuse, and at first may be noticed only on the face and hands. There is sometimes associated with it an opposite condition in which the normal pigment is absent—leucoderma. The mucous membranes of the lips, mouth, conjunctivæ, and vagina show evidences of abnormal pigment deposition. The usual view, that the conjunctivæ sclerarum are always free, is untenable, as not infrequently little areas of intense pigmentation may be seen there.[1] It has been noticed by many that the hair of patients suffering from the affection grows gradually darker during its progress. The accompanying colored plate is from a case which died in the Montreal General Hospital.[2]

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[1] *Zeigler's Beitr. z. path. Anat.*, 1889.

[2] *Beiträge*, 1891.

Disturbances of digestion are among the most common symptoms; apparently causeless nausea and vomiting may occur early; the vomiting may be paroxysmal. Diarrhœa without any apparent cause is frequent. The circulation is depressed and the heart's action feeble; the pulse small and compressible. The hands and feet may feel cold and clammy—a condition which is a source of much discomfort to the patient.

Severe fainting spells, causing much alarm, and which sometimes terminate fatally, are not rare. Addison laid considerable stress on an accompanying anæmia, but this is by no means a constant symptom, for in a good proportion of the cases the number of red blood-corpuscles is normal. There is no increase in the number of white cells. The presence of free pigment-granules in the blood has been described.

The asthenia is usually well marked, and apparently out of all proportion to the general condition. There is disinclination for all kinds of exertion, and the slightest mental or physical effort causes fatigue. The face has an expression of weariness, especially in the more advanced stages of the disease. The prostration may finally be so extreme that the patient is compelled to remain in the recumbent posture; the voice grows weak and small, the intelligence becomes dulled, and in some cases there is delirium. Pain and tenderness are present in the lumbar and epigastric regions in about one-third of the cases.

On account of the evidences of disturbed tissue-metabolism many analyses of the urine have been made in Addison's disease in the hope that the results would throw some light upon its nature. The quantity passed is in the majority of cases not far from normal, and, although in some instances it is diminished, in others there seems to be a distinct polyuria. Fleischer suggests that the former may indicate irritation, the latter destruction of the adrenal glands. As a rule, there is no albuminuria. Many observers have noted an increase in the amount of indican, as shown by the reaction of Jaffé; and this we might have expected, since we know that indicanuria is frequent in nearly all cachectic diseases associated with destruction of albuminoids. Nothing can be positively said about the amount of urea excreted, although Thudichum and Rosenstirn thought it was decreased. The volatile fatty acids appear in the urine in amounts considerably above normal, and there would seem to be some diminution in the quantity of kreatinin given off. Urobilin and uromelanin may be present in excess.

Diagnosis.—We must not conclude that in every case where we find patchy pigmentation we are dealing with Addison's disease. There exists a whole series of conditions which may give rise to an increased pigmentation. In abdominal growths, tubercle, cancer, and lymphoma there may be extensive pigment-deposition on the face or other parts, and I have frequently observed this phenomenon in tuberculous peritonitis. Diseases of the uterus and of its adnexa are not infrequently accompanied by a patchy melasma, and the discoloration of the face which occurs in many pregnant women—the "*masque des femmes enceintes*"—is well known to all. Certain diseases of the liver (*e. g.* diabetic cirrhosis) may induce pigmentation of the skin, so that the popular idea of "liver-spots" is not altogether devoid of basis. In persons who are overworked and allow themselves to become constipated discolorations of the skin may occur, and we should bear in mind the possibility of the existence of melanotic cancer somewhere in the body, as this sometimes gives rise to marked staining of the skin. Drummond has noted a melasma in cases of Basedow's disease.

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[1] Leva, *Virchow's Archiv*, Bd. cxxv. p. 35.

[2] *Trans. of the Canada Med. Association,* 1877.

The diagnosis in the negro would be extremely difficult, not only because the skin is normally black, but also because there are not infrequently found dark patches on the mucous membrane of the mouth in this race even in health, of which the discoloration seen in the so-called "blue-gummed" negro, whose bite is popularly supposed to be poisonous, is an example.

It would scarcely be necessary to mention those cases of vagabond's discoloration where there are marked pigment-deposits resulting from an old irritation produced by lice and dirt, had they not before now been mistaken for signs of Addison's disease.

In every case of abnormal pigmentation we must endeavor to decide which of these various possible conditions is the cause, and the diagnosis of morbus Addisonii is scarcely justifiable unless there be at the same time distinct evidence of asthenia and of gastric irritability. Painful sensations sometimes occur in the region of the kidneys, but these are of no help in making the diagnosis. The cases running a rapid course with severe prostration and digestive disturbances, when not accompanied by discoloration of the skin, cannot be positively diagnosed during life. The disease has at times resembled typhus so closely that the mistake was only found out at autopsy.

Prognosis.—The disease is in every case fatal. Acute cases run their course in a few weeks; its average duration is about one year, but Schmaltz has recorded the case of a patient who lived for ten years after the first appearance of the symptoms. Distinct remissions may occur during which the patient enjoys comparatively good health. Death usually results from the asthenia, but toward the last there may be convulsions and coma.

Treatment.—From what we have before said it will be understood that the treatment can at best be only symptomatic. We are powerless to check the advance of the local mischief. Counter-irritation by means of the cautery or by strong iodine applications have been advised and may be tried. The dangers of syncope must be clearly put before the patient, and he must be advised to lead a quiet life, so that both mind and body shall have as much rest as possible. I remember a professional man who consulted me with regard to bronzing of the face and hands; he had only one fainting spell. On account of his general good condition and the limited character of the bronzing it seemed possible that it might not be a case of Addison's disease, and he was advised to give up business for a year and live quietly abroad; but while making arrangements to follow out this advice he had another sudden attack of syncope which proved fatal.

General tonic measures must be employed to support the patient's strength, and when there is any aggravation of his asthenic symptoms he should be strictly confined to bed; at these times stimulants may be indicated. If there be anæmia, iron in some form should be given in large doses. Arsenic, strychnine, and phosphorus may be used for their tonic effects.

The treatment of the digestive disturbances is very important, and to meet them the physician's skill will be taxed at times to its utmost. The diet should be light and nutritious, many patients doing best when confined to a strictly milk diet. For the nausea and vomiting we would recommend creasote, hydrocyanic acid, iced champagne, etc. On account of the liability to profuse diarrhœa great caution must be used with regard to purgative medicines, and constipation when it exists is best relieved by enemata. If there be diarrhœa, bismuth in large doses acts well.

It is impossible at present to say whether the use of the extract of the glands will prove curative.

Diseases of the Thyroid Gland.

(1) Congestion.

A marked feature of the thyroid gland is its rich vascular supply, the arteries being estimated by Soemmering as proportionately eight times larger than those of the brain. A transient increase in the amount of circulating blood, to which the term congestion should be limited, is of interest in connection with certain physiological processes, and as a preliminary step in the production of one form of goitre. Transient enlargement of the gland occurs sometimes at puberty, with the establishment of menstruation. Abrupt suppression of the menses has been followed by acute goitre. Sexual excitement in women (and in animals) may be associated with congestion of the gland. To establish a bride's virginity an old custom recommended the measurement of the neck before and after defloration (*Non illam genitrix orienti luce revisens hesterno poterit collum circumdare filo. Catullus*). That engorgement of the gland followed conception is an idea which dates from Democritus, and in reality the condition is met with in a considerable number of pregnant women.

A congestion leading to a fulness of the gland has been noticed also in certain of the infectious diseases, particularly in small-pox and typhoid fever, and it is stated to occur occasionally during the paroxysm of malarial fever. Repeated attacks may precede the development of goitre.

The symptoms are trivial, amounting only to slight bilateral swelling, usually with marked throbbing of the arteries. In a previously healthy gland the congestion rarely attains a grade sufficient to induce serious consequences, but there are instances of hæmorrhage into the gland and of slight dyspnœa. The cases of extreme engorgement with serious symptoms have occurred usually during pregnancy.

(2) ACUTE INFLAMMATION OF THE THYROID (THYROIDITIS).

This may develop either in a previously healthy gland or in one affected by goitre, in which case the term *strumitis* is used by German writers. Trauma, cold, and the rheumatic poison are assigned as causes, but probably in the great majority of the cases the inflammation is associated with one of the infectious diseases, more particularly variola, typhoid fever, typhus fever, or malaria. The acute inflammation of the goitrous gland—strumitis—is also very rarely primary, but is commonly a metastatic affection in the course of some febrile disorder. The streptococcous infection is most frequent, but the pneumococcus and the bacterium coli commune have also been found (Tavel).

The acute thyroiditis is manifested by the local symptoms of the inflammation in one or other lobe of the gland (rarely median) and the constitutional disturbances accompanying fever. The swelling may be very great, and death has been caused by compression before suppuration has occurred. Resolution occurs in a number of cases, but in a majority the inflammation issues finally in suppuration. If opened, recovery takes place rapidly. Sometimes the thyroid cartilage is denuded. Rare and very serious terminations are burrowing of the abscess behind the trachea with inflammation and compression, and rupture of the abscess into the air-passages or into the œsophagus. Gangrene has been described as a sequence of dissecting thyroiditis.

The recognition of acute inflammation of the thyroid is easy on account of the swelling and redness in one or the other lobe. In typhoid fever it must not be mistaken for laryngo-chondritis, which may produce swelling and redness in the front of the neck just above the thyroid.

(3) GOITRE (BRONCHOCELE; STRUMA).

With the involvement of the follicular, fibrous, and vascular tissues respectively, Virchow recognized *follicular*, *fibrous*, and *vascular* forms of goitre; while other varieties, dependent upon special changes, are the amyloid, cystic, colloid, and calcareous.

Follicular or parenchymatous goitre, the most common form, represents a hyperplasia of the follicles of the gland, often in definite peripheral nodules, but also in the deeper parts of the lobes.

In the fibrous goitre, in addition to the follicular hyperplasia, there is enormous increase in interstitial tissue, which leads to induration. When of long duration the consistence of this variety may be that of cartilage, and cystic degeneration is common.

Even in the parenchymatous form the blood-vessels are abundant, but in other instances the blood-vessels are greatly increased in size, the arteries as well as the veins. In the so-called aneurismal goitre the arteries are chiefly involved, and in extreme cases the whole gland is converted into a spongy erectile tissue, which expands forcibly with each cardiac systole. In the other and more common variety the veins are enlarged; it is usually a complication of the ordinary follicular struma. The dilated veins are chiefly at the periphery of the gland, but in extreme cases the deeper vessels are greatly enlarged and sacculated. Special varieties result from changes and degenerations in one or other of these forms. The amyloid and colloid changes are common. More important is the development of cysts in the large goitres, the contents of which are variable, sometimes containing the colloid material of the gland, at others a brownish, coffee-colored, dark liquid containing cholesterin, fatty débris, and altered blood. In very old, fibrous goitres the calcareous degeneration is common.

Etiology.—Goitre occurs, first, as a sporadic affection; second, as an endemic form, in which, owing to local conditions, a considerable number of individuals in the community are subjects of the disease; third, epidemic goitre, in which form within a short space of time a considerable number of individuals in a certain region are attacked with acute swelling of the thyroid gland; and lastly, goitre is one of the chief features in the affection known as Basedow's disease or exophthalmic goitre.

Sporadic goitre occurs in all parts of this country, and in a majority of the cases it is impossible to assign any definite cause for the swelling of the gland. Heredity is met with in a few cases; in others, disturbance of the sexual functions.

Endemic goitre occurs chiefly in mountainous districts, and is found particularly in regions of the Alps and Pyrenees. In this country there is no very recent information as to its occurrence in endemic form. Benjamin Smith Barton, in his valuable monograph (1800), speaks of its prevalence in certain portions of New York State, among the Canadians in Detroit, and in the district of St. John in Lower Canada. Cases were formerly numerous in certain sections of the Green and White Mountains. In the limestone region about Kingston, Ontario, the disease is very prevalent, and Dr. Clark of the Kingston Asylum writes me that fully 50 per cent. of the cases in the asylum have bronchocele. The disease is so prevalent in this district that certain strains of dogs and horses are regularly goitrous. In reply to inquiries in those towns of Vermont, New York, and Alabama in which the disease was stated to be endemic in the early years of this century, the physicians state that goitre no longer prevails to any special extent.

Much discussion has taken place as to the essential cause of endemic goitre, but no one of the theories meets all facts of the case. In the infected regions heredity plays an important rôle, and children have been born with the gland enlarged. The affection would appear to be in some way associated with the drinking water, but there is no unanimity of opinion as to the nature of the impurities which produce the disease. The water of limestone districts appears to be most prone to cause the disease.

Epidemic goitre has been met with chiefly in certain parts of Europe, particularly in goitre districts. Regiments of troops transferred to a certain garrisons in these regions have become within a short time subject to the disorder. Within a month after the arrival of the troops in an infected region 30 or 40 per cent. of the number may have enlargement of the thyroid. The facts of this remarkable disorder have made many observers regard goitre as an infectious malady due to an unknown poison which requires some local conditions for its development. Details of these epidemics are given in Hirsch's work on *Geographical Pathology.*

Symptomatology.—In a majority of cases the disfigurement is the sole complaint, but serious symptoms may result from pressure of the tumor on the trachea or on the veins. The large growths are not so likely to cause pressure as the smaller ones which pass beneath the sternum or which encircle the trachea and produce narrowing of its lumen with a distressing dyspnœa. Much more rarely the œsophagus is compressed by the enlargement of a peripheral portion of the gland. Pressure on the veins of the neck may impede the return of blood from the brain and cause headache and drowsiness. Instances also have been reported of compression of the carotids by the tumor, and marked cerebral symptoms, such as tetany or convulsions. Serious pressure on the nerves is uncommon.

And, lastly, goitre may be accompanied with profound changes in the general nutrition and in the cerebral functions, leading in the child to the condition of cretinism, in the adult to myxœdema. These changes would appear to occur only in instances in which the function of the gland is totally abolished.

The recognition of goitre is usually easy, and the condition is distinguished readily from tumors in the front of the neck, such as congenital, sebaceous, and blood-cysts, abscesses, malignant growth of the gland, and lymphadenoma.

Aberrant portions of thyroid tissue are found from the hyoid bone to the arch of the aorta, and in most remarkable situations, such as in the hyoid bone itself and in the trachea. Sometimes these structures develop into large tumors which from their situation may be difficult to recognize. Aberrant portions may form large, intrathoracic growths. I have reported an instance in which at the top of the left pleural cavity, outside the pleura, there was a tumor as large as an orange, the anatomical characters of which left no doubt that it had developed from an aberrant thyroid. Dettrich has reported an instance in which the tumor from an aberrant portion of the gland formed a cystic tumor of the size of a man's head, which occupied the greater part of the right side of the chest.

Treatment.—In districts in which goitre prevails individuals should drink only boiled water. Early removal to a non-goitrous district is followed, as a rule, by disappearance of the tumor. Of medicinal remedies, iodine internally, in the form of iodide of potassium, 10 to 20 grains three times a day, with the external application of iodine, is sometimes beneficial. A large number of remedies, internal and external, have been advised, the great majority of which are useless. In early cases of the soft, follicular form active counter-irritation with iodine, externally, should be employed. The biniodide-of-mercury ointment is much lauded. In some instances good results have followed the internal administration of ergot. In the long-standing cases of large fibrous goitres, internal remedies are useless, and surgical treatment should be resorted to, either injections of various solutions. ligature of the arteries, or removal of large portions of the gland.

(4) MYXŒDEMA.

Definition.—A constitutional disorder consequent upon the loss of the function of the thyroid gland, characterized by an infiltrated (myxœdematous) condition of the subcutaneous tissues, and in children by arrest of development; in adults, by the production of a cretinoid state.

In 1873, Sir William Gull reported cases under the title of "A Cretinoid State supervening in Adult Life in Women." Dr. Ord in 1877 grouped these cases under the term *myxœdema*, and discussed the relations of the condition to atrophy of the thyroid and to epidemic and sporadic cretinism. Kocher had noticed that in certain instances of total extirpation of the thyroid gland a remarkable cachexia developed (cachexia strumipriva), which in reality is identical with the condition described by Gull and Ord. Horsley demonstrated experimentally that loss of the function of the thyroid gland in animals produced symptoms identical with those of myxœdema and the cachexia strumipriva. As suggested by Ord, endemic and sporadic cretinism are really affections associated with a similar loss of function of the thyroid gland.

Practically, three groups of cases may be considered:

Cretinism, Sporadic and Endemic.—The loss of function of the thyroid gland may be either due to complete atrophy, or it may be enlarged and the function abolished by the substitution of fibrous tissue for normal gland-structure. The condition which results when this exists congenitally or develops in early childhood is known as cretinism. The congenital cretin rarely survives birth. The body is stunted, broad, and the subcutaneous tissues much developed; there are marked changes in the skeleton. The thyroid gland is either completely atrophied or congenitally absent. In other instances the child shows very slight traces at birth, but as development proceeds the condition becomes very manifest; there is subcutaneous swelling due to myxœdema, the head is large, the neck short and thick, the lips full, and the tongue large and thick. The intelligence is variable, but the mental development is extremely slow. In many instances there is slight enlargement of the thyroid gland. In some cases there is a definite goitre. In other cases, again, the condition does not develop until from the third to the fifth year, up to which time the child may have thriven and have been quite natural. The growth is retarded; the head becomes disproportionately large and broad, the nose retrousée, the lips thick; the limbs are short, the subcutaneous tissues infiltrated; the skin has a glossy appearance, and there is a thickened, solid œdema above the clavicles. The voice has either a rough or a stridulous character, and the hair often becomes thin and scanty. This change is usually progressive, and reaches its maximum between the twelfth and fifteenth years. The child never properly matures, and, the condition remaining stationary, at the twentieth, or even the thirtieth, year the mental and physical characters are those of childhood. The change in the thyroid gland is either progressive atrophy or the development of a goitre which interferes with its function.

Cretinism prevails extensively in regions in which goitre is endemic, and the cases of congenital and early cretinism are usually in the children of goitrous parents. Sporadic cases occur in all countries. They are rare in America. I have been able to collect about a dozen cases after extensive inquiries. Cretins are grouped with idiots, but the use of the term "cretinoid idiocy" should be carefully restricted to such cases as those above described, and associated with definite changes in the thyroid gland and in the skin and connective tissues of the body.

Myxœdema Proper.—This, the cretinoid state of Gull, develops in adult life in consequence of abolition of function of the thyroid gland. The main facts with reference to it will be found in the report of the Clinical Society of London for 1888, from which the following description is culled: The disease is more frequent in women than in men in the proportion of 1 to 6. It may affect several members of a family, and may be transmitted through the mother. Neuroses have been present in many instances or have preceded the symptoms—thus, cases have developed in connection with exophthalmic goitre. The disease has apparently no relation to disturbed catamenia or to pregnancy, though in one instance there was a remarkable alternation of the condition of myxœdema with pregnancy, the entire swelling passing away, to return after the birth of the child, the phenomenon recurring three times during seven years. A transient myxœdematous swelling of the face and of the hands may occur in connection with the acute development of a goitre, and disappear with its subsidence, and a transient myxœdematous condition is met with in some instances of exophthalmic goitre. Myxœdema does not appear to be so common in America as in England.

The following are the main symptoms as described by Dr. Ord in the report of the Clinical Society: "Under the head of type-signs the following may be enumerated: The marked increase in the general bulk of the body; the firm swelling of the skin, not pitting on pressure, inelastic, adherent to the parts beneath, and not affected by gravitation; the dryness and roughness of the skin, tending, with the swelling, to obliterate all lines of expression; the imperfect nutrition of the hairs, leading to their loss; the local tumefaction of the skin and subcutaneous tissue noticed in various parts of the body, but most frequently in the subclavicular regions; the turgescence and infiltration of mucous membranes, leading, in the mouth, to an affection of the teeth homologous with that of the hairs just mentioned; the remarkable physiognomy; the slow, painful utterance, monotonous voice, and leathery quality of tone thereof, with curious nasal explosions at short intervals during speaking; the slowness of thought and movement; the slowness of perception and response; the defect of memory; the frequent occurrence of mental disorders of an irritable and suspicious character, or of hebetude and somnolence alternating with excitability; the tendency to fall, owing to disorder of co-ordination; the existence of subnormal temperature of the body; the aggravation of all symptoms during low climatic temperatures; and the diminution or apparent absence of the thyroid gland. Among the minor or accessory signs may be quoted abnormal subjective sensations, belonging particularly to taste and smell; occipital headache; marked alterations of temper; and a curious persistence of thought and action, overriding all attempts at interruption by friends or observers."

In nearly half the cases delusions and hallucinations occur chiefly in the advanced period. Muscular tremors and contractures have been described. Hæmorrhages are not uncommon, chiefly bleeding from the nose, gums, teeth, and bowels. The course of the disease is slow, but progressive. Death usually occurs from intercurrent disease. Lately, in many cases, the disease has been arrested, or even cured, by means which will be referred to later.

Operative Myxœdema (*Cachexia Strumipriva*).—Complete extirpation of the thyroid gland is followed in men and animals by the gradual production of a bodily and mental condition identical with that of myxœdema, and to which Kocher, who first noted it, gave the name cachexia strumipriva. The experiments of Horsley and others have shown that if a very small portion of the gland be left these symptoms do not occur, and the cases of total extirpation in animals which survive have been those in which accessory glands have been present. These have been found in all parts, from the base of the tongue to the aorta, and thyroidal tissue has even been found in the hyoid bone and in the trachea.

Horsley has shown that complete removal of the thyroid gland in monkeys induces myxœdema, and if the animals are kept warm a state ultimately develops which closely resembles cretinism. In man the disease follows only a certain number of total, and a much smaller proportion of partial, removals of the gland. Of 408 complete thyroidectomies analyzed in the Clinical Society's report, in 69 myxœdema developed.

These three conditions, then—cretinism, myxœdema proper, and operative myxœdema—not only stand in close relation to each other, but are in reality identical affections induced by the loss of the function of the thyroid gland, which would appear to supply some material necessary to the maintenance of the normal metabolism, more particularly to that of the connective tissues of the body.

The **diagnosis** is rarely difficult. The firm, inelastic subcutaneous thickening, the dry, rough skin, the supraclavicular swellings, with mental dulness or defects, occurring in connection with atrophy of the thyroid gland or following its extirpation, could scarcely be mistaken for any other disorder.

Treatment.—Until recently this was a question of palliation only. As the patients suffer extremely from cold, they should be kept at an even temperature, and, if possible, should spend the winters in a mild, equable climate.

Repeated warm baths, shampooing, Turkish baths, hot electric baths, and pilocarpine hypodermically may be employed. Arsenic and strychnine are also recommended. Recently very brilliant results have been obtained in many cases by the employment of extract of the thyroid gland, which may be given in many different ways. The fresh thyroid of the sheep or the calf may be taken either as a watery extract or glycerin extract, or the fresh gland may be finely minced and taken raw or lightly broiled. The glycerin extract or the watery extract may be used hypodermically, and special preparations have been made by many chemists. From a quarter to half a gland may be taken daily. It is well to begin with small doses and increase gradually; very serious symptoms have followed the administration of too large quantities at short intervals. Most remarkable changes have followed its use in cases of myxœdema, and not only has the physical condition returned almost or completely to normal, but the mental symptoms have disappeared. Similar good effects have been obtained in cases of sporadic cretinism.

DISEASES OF THE THYMUS GLAND.

Disorders of this gland are rare. It is important to remember two anatomical facts: First, that the size of the gland may vary greatly in infants, and even glands weighing as much as five hundred grains have been found without any special symptoms; and second, that the organ, instead of undergoing the physiological atrophy, may persist in the adult and form a prominent mass in the anterior mediastinum.

Hypertrophy of the organ, referred to by many writers, is difficult to determine on account of the extreme variability in its size. Formerly it was believed that enlargement of the gland produced an affection known as thymic asthma, but recent writers have failed to associate either an asthmatic disorder or the spasm of the glottis with any condition of the thymus. On the other hand, there are some instances of sudden death in children in which marked enlargement of the thymus has been found. Jacobi in his monograph on the thymus quotes two cases from Grawitz: "One was that of a child of eight months who was found dead in bed after having been in perfect health. At the post-mortem examination absolutely nothing was found to explain death except a thymus of unusual size, which was flattened and covered the larger part of the pericardium, and extended upward to an unusual degree in the direction of the thyroid gland. The second was a babe of six months, in perfect health, which, while being carried on the arm of the father, was taken with an attack of dyspnœa, became cyanotic, and died in a few minutes. At the post-mortem it was found that there was a large amount of subcutaneous fatty tissue and symptoms of rachitis about the chest. The thorax was broad and the abdomen somewhat inflated. The diaphragm reached upward to the fourth rib on both sides. The thymus was very large, its two lobes covering the larger part of the pericardium, and two processes reached upward to the thyroid gland. Longitudinally it measured seven and a half centimetres; over the pericardium it was more than six centimetres wide; its thickness was one and a half centimetres, with the exception of the region of the manubrium sterni, where the dorso-ventral diameter amounted to one and four-fifths centimetres. The tissue of the thymus was of a grayish-pink color, quite firm, and contained a great many punctate hæmorrhages. There was a large amount of blood in the heart and the two venæ cavæ. The epiglottis was compressed from the two sides. The spleen was large, and the mesenteric glands were larger than normal."

Hæmorrhages are occasionally met with, but are apparently of no moment.

Inflammation of the gland is described by a few writers, but it is an ill-defined affection, not to be recognized during life, and many cases have undoubtedly been confounded with inflammation of the mediastinum and of the lymph-glands.

Syphilis is more common, either in the form of a definite gumma or as a connective-tissue proliferation, and there are cases of markedly syphilitic infants in which abscess-cavities have been found in the gland, or cysts containing a clear or yellowish fluid.

Tuberculosis may occur as an isolated, primary lesion, as in the case of Demme, but more commonly as miliary tubercles or cheesy areas in cases of general tuberculosis in infants.

Of neoplasms, sarcoma, cancer, lymphadenoma, and myxolipoma have been described, and the thymus has been found involved in a few instances of leukæmia. Lastly, Jacobi has noticed in diphtheria changes in the cells similar to those described by Oertl in the lymph-glands in this disease.

DISEASES OF THE PERICARDIUM.

BY WILLIAM PEPPER.

PERICARDITIS.

Definition.—Pericarditis is an acute, subacute, or chronic inflammation of the pericardium, characterized by fibrinous or fluid exudation or the formation of fibrous adhesions.

Etiology.—Pericarditis may be primary or secondary according as it is an independent affection or consequent upon an existing disease.

Primary pericarditis is much less common than secondary. The most frequent cause is exposure to cold. This form of idiopathic pericarditis from exposure has been the subject of no little difference of opinion, and many authorities even deny its existence, but undoubted cases have occurred, and I have seen instances where no other explanation of the pericarditis was possible. Very probably, however, in many of the cases reported, especially in children, in whom this form is most frequent, a masked rheumatic affection, articular or tonsillar, has escaped observation. Traumatism, as blows or crushes or penetrating wounds, is an occasional cause. Very interesting, though extremely rare, are the cases in which the injury has resulted from the lodgment of a foreign body, as a small bone or a plate of false teeth, in the œsophagus. Tuberculosis of the pericardium, more frequently secondary, may be primary, and generally occurs as a part of a general primary tuberculosis of the serous membranes, though the pericardium alone may be involved.

Secondary pericarditis may be the outcome of some general bodily condition which predisposes the individual, or it may be the direct result of the primary disease, the latter being more common. It may result from direct extension of inflammation in cases of pleurisy, particularly in pleuro-pneumonia, of inflammatory diseases affecting the lymphatic glands of the mediastinum, the adjacent bones, the œsophagus, or even the abdominal organs, as in abscess of the liver. Direct extension of inflammation from the heart wall in myocarditis, and from the valves in valvulitis, particularly aortic, has been considered a common cause; and Fagge found more than half of his cases of fibroid heart complicated by pericardial disease. More frequently, however, in such cases the myocarditis is the secondary disease. Of the general diseases to which pericarditis is secondary, the most common are acute rheumatism, Bright's disease, tuberculosis, and the infectious fevers; more rarely it occurs in diseases which profoundly affect the general nutrition, such as scurvy, diabetes, and alcoholism. Gout also has been reckoned among the causes, but generally the associated nephritis is operative in this disease.

By far the most important cause of pericarditis is acute articular rheumatism. In the elaborate statistics of Sibson pericarditis occurred in 63 of 326 cases, and even this proportion is probably too low. As a rule, pericarditis occurs during the first or second week of the rheumatic attack, but may be found at the very onset, and in a small percentage of the cases actually precedes articular involvement. Tonsillitis and chorea manifest their rheumatic nature in some cases by the occurrence of pericarditis during their course—less frequently, however, than by endocardiac disease.

Next to rheumatism in point of frequency comes Bright's disease, but in this case pericarditis is a late complication, and often it is the terminal disease. The chronic interstitial form is most frequently the cause; amyloid disease is rarely so. In any case the supervention of uræmia is apt to determine the development of the pericarditis. Tubercular infection of the pericardium, though occasionally primary, usually results from neighboring tuberculosis of the lungs, pleuræ, lymphatic glands, or bones by direct extension, or from more remote tuberculosis by dissemination through the blood. Of the acute infections, pyæmic diseases, as puerperal fever, scarlatina, and influenza, are the more common; less frequently small-pox, measles, or typhoid fever is the primary disease. Croupous pneumonia also may be counted as one of the causes operating through hæmatogenous infection, though, as before stated, it more frequently leads to pericarditis by extension of inflammation from the accompanying pleurisy.

Pericarditis may occur at any age, but it is more common in young adults between twenty-five and thirty-five than in children or the old. Cases in which the disease occurred in fœtal life have been recorded, and Behier observed an instance of adherent pericardium in an infant of eleven months. The idiopathic form seems most common in children, as are also the cases due to infectious diseases; the rheumatic is the usual variety in young adults, while in patients beyond middle life interstitial nephritis is frequently the cause. Sibson found that the sexes are equally affected before the age of twenty-one years, but after that age males are far more liable. Surrounding conditions leading to great exposure and cold climate aid in the development of pericarditis, doubtless by furthering the diseases upon which it depends.

Morbid Anatomy.—The morbid changes found in the pericardium are nearly always diffuse, affecting the greater part of both visceral and parietal reflections; but in some cases a very limited area of inflammation is seen anteriorly on the surface of the right ventricle, posteriorly on the auricles, or at the base or over the roots of the great blood-vessels. The first change seen on the surface of the pericardium is injection of the blood-vessels and a lustreless appearance of the membrane. This soon gives way to exudation of fibrinous material, so that when seen at the very earliest time there is usually a thin whitish coating, which may be stripped from the tissue beneath. Later, the exudation increases in amount, and by the constant attrition in the cardiac movements a shaggy or hairy appearance is produced, which has been designated by old writers as the *cor villosum.* The fibrinous deposit may attain a thickness of a quarter to half an inch. With this plastic exudate there is nearly always a small amount of serous fluid within the fibrinous meshes, and generally in the later stages considerable fluid exudation takes place, distending the pericardial sac. The fluid exudation consists of serous, sero-fibrinous, purulent, or hæmorrhagic liquid, and varies in amount from 100 c.c. to a litre or two. The rapidity of the effusion varies in different cases, but usually it reaches the maximum in two to four days, and the re-absorption occurs in the course of a week or ten days. The exudation may, however, be very slowly absorbed or remain as a chronic condition. The character of fluid found depends upon the cause of the pericarditis; usually being serous in idiopathic and rheumatic cases; purulent in cases secondary to pyæmic diseases, where the pericarditis has resulted from extension of inflammation from empyema, suppurating lymphatic glands or necrosis of bone, or where infection has been carried by a trocar in tapping a serous effusion. I have been struck by the frequency of purulent pericarditis in cases of influenza, and in tuberculosis the same is often observed. Hæmorrhagic effusion occurs in scorbutic or purpuric cases, in the old and cachectic, but especially in tuberculous and cancerous pericarditis.

After resorption of the fluid, and in cases in which the effusion of fluid did not take place, the fibrinous exudation may be completely absorbed. In other cases the adjacent layers of the pericardium are agglutinated by the exudation, and later united by fibrous adhesions, the result of connective-tissue hyperplasia. Such adhesions may be short and general, leading to a universal adhesive pericarditis, or there may be but a few long bands, especially at the apex, where the constant motion tends to elongate them. Purulent effusion is of course less likely to be absorbed, but the evidence of partial absorption is sometimes found in the discovery at autopsy of greatly thickened and cheesy deposits upon the surface of the membrane. Subsequently in such cases limy infiltration may occur within the deposit, and I have seen an instance in which the heart was completely encased in calcareous envelope. Not infrequently there may be seen upon the surface of the pericardium whitish "milk-spots," which are areas of hyperplasia of the endothelial lining and subendothelial connective tissue, the result of previous inflammation.

The study of the micro-organisms in pericarditis has thus far been unsatisfactory, though pyogenic bacteria, the pneumococcus, and tubercle bacillus have been detected.

The most important secondary effects of pericardial inflammation and exudation are found in the heart. In nearly all cases there is a certain amount of myocarditis affecting the outer portions of the heart-muscle to the depth of a few millimetres; in more intense inflammations the myocarditis is deeper and more general. In cases in which there is a large chronic effusion the pressure of the liquid upon the heart and great vessels leads at first to a hypertrophy of the heart-walls, and later in some cases to atrophy, the result of obstruction to proper nutrition of the muscle. Great hypertrophy is also found in adhesive pericarditis. Many authors hold that this is due to the associated valvular disease which is so common, but I have seen extensive hypertrophy without valvular disease, and am disposed to regard the adhesions as the important cause. The cardiac enlargement in pericarditis is in part due to myocardial inflammation. Finally, fatty degeneration and dilatation are apt to occur as late results of improper cardiac nutrition. In cases of purulent effusion the heart may be involved by purulent myocarditis; and in very rare instances a purulent myocarditis ruptures externally, inducing secondary purulent paricarditis

Symptomatology.—The onset of pericarditis is rarely marked by decided symptoms, and may be so insidious that the disease escapes detection. It is not unusual to find at autopsy evidence of extensive acute pericarditis in which no clinical manifestations of the disease had been present.

There may be rigor or a decided chill at the onset, with gastric disturbances, and slight pain, when present, is a suggestive symptom, but is rarely severe and frequently is absent entirely. The pain is largely due to attrition of the inflamed pericardial surfaces, and therefore to some extent depends on the degree of inflammation present. In not a few recorded instances pain has been truly anginal, sharp and lacerating in character, and radiating to the left shoulder and down the arm. In cases in which pain is absent there may be a vague sense of distress, or there may be decided tenderness on pressure over the præcordia or in the epigastric region. In rare cases all abnormal sensations over the heart are absent, but there is pain posteriorly when the patient swallows food, eructates, or vomits. As the disease progresses and fluid distends the sac pain may disappear, but this is not always the case, and sometimes it grows more severe. It is evident, then, that the pain is not wholly due to friction.

The fever in uncomplicated cases rarely exceeds 102.5° F. It is irregular in course, often presents marked remissions, and finally disappears by gradual lysis. Cases of rheumatic pericarditis with extreme hyperpyrexia and marked cerebral symptoms have frequently been reported; but in these cases the temperature cannot be ascribed solely to the pericarditis. With the rise of temperature there is a coincident acceleration and fulness of the pulse. In the early stages it is regular, though occasionally sudden irregularity and rapidity of the pulse may be the first indication in rheumatism of the pericardial complication. Later, as the effusion of fluid increases and pressure is exerted upon the heart and great vessels, the pulse loses decidedly in fulness and force, and becomes small, weak, and irregular. Dyspnœa is a common symptom, and at times the sense of suffocation is intense and orthopnœa appears. In the early stages rapidity of breathing is due to cardiac excitement and reflex nervous disturbance, but in the later stages the pressure of the effusion upon the bronchi and lungs is a potent factor. The patient then sits up in bed or inclines to the left side, and in some cases finds comfort only by lying completely prone, so as to relieve the pressure exerted by the fluid on the bronchi and lungs.

With increasing effusion pressure-symptoms of various kinds may make their appearance. As stated, the heart becomes weak and irregular, the respirations are hurried and difficult; there may be hiccough, nausea, and vomiting from the weight of the fluid upon the diaphragm, and pressure upon the roots of the large veins may cause fulness of the cervical veins or even the superficial veins of the thorax. The lips and finger-nails are blue, the skin becomes of a cyanotic color, but usually at the same time pale—pallid cyanosis. Pressure upon the œsophagus causes dysphagia; the left lung is more and more compressed, and dyspnœa increases; the left recurrent laryngeal nerve as it curves around the arch of the aorta may be pressed upon, and aphonia result; very rarely both recurrent laryngeal nerves have been involved, with bilateral palsy of the vocal cords.

With increasing cardiac failure and venous stasis cyanosis grows most marked, and dropsy, sometimes universal, more commonly in the feet and in the pleural and other serous cavities, supervenes. At this time the slightest physical exertion, as sitting up in bed, may cause sudden heart-failure and death.

The nervous symptoms in pericarditis are most important, but, like hyperpyrexia, they are more often the outcome of the primary rheumatic or other disease than the pericarditis. Late in a case with failing circulation and congestion of the brain, headache, dulness, apathy, and stupor are frequent symptoms. Active delirium, maniacal excitement, coma, convulsions, and chorea have all been noted as symptoms, especially in cases attended with hyperpyrexia; and, though all of these may be found in rheumatism without pericarditis, they are apt to be of greater gravity when this complication is present, and occasionally occur in non-rheumatic pericarditis. Melancholia with hallucinations was particularly described by Sibson.

Physical Signs.—The description of pericarditis as of two stages, the dry or plastic and that with effusion, has an especial value in the study of the physical signs. During the first stage the friction of the two layers of the pericardium upon each other gives rise to distinctive signs. On palpation over the heart, besides the slightly increased impulse, there may be felt a peculiar rough friction-fremitus. This is most distinct when the inflammatory process is intense and the chest-wall thin, but it is by no means a constant physical sign. It is generally grating or rough in quality, gives the impression of being close beneath the hand, and is limited to the area of impulse and in duration to the time during which the impulse is manifested. A more constant and more important sign is the auscultatory friction-sound. The characters of this sound are exactly those which we should expect from its causation. It is harsh, grating, and nearly always double, to-and-fro, corresponding roughly with the revolutions of the heart. More rarely it is a soft sound, not unlike an endocardial murmur, and it may be single or even triple. In some cases the sound is peculiarly creaking, to which the name of "new leather" friction has been appropriately applied. The heart-sounds may be heard with or through the friction-sound, but do not correspond exactly in time—as a rule outlasting it. When the friction is very distinct it may almost completely obscure the sounds of the heart. The friction is made more intense by firm pressure of the stethoscope, by full inspiration, or by the erect posture of the patient. I have in several cases succeeded in making the diagnosis only by observing a decided friction-sound on heavy pressure with the head in direct auscultation, when with little pressure or with the stethoscope alone the sound could not be detected. The position in which the friction is best heard varies in different cases, and in the same case when effusion begins to form. It is usually detected along the left border of the sternum, over the right ventricle, or over the base of the heart, more rarely at the apex. When effusion begins to accumulate and the heart is pushed upward, the sound also is heard farther up and to the right, and when the sac is fully distended with fluid, the friction usually disappears, though I have seen a number of cases in which a rub was audible at the roots of the great vessels throughout a case with marked effusion.

The signs indicative of pericardial effusion are more marked than those of plastic pericarditis. On inspection, besides the general features of the case, the distended cervical veins, the blue lips, and cyanosis, there are important local indications. In children the softness of the cartilages and ribs and the thinness of the chest-wall permit of considerable bulging in the pericardial region, and even between the costal cartilages; in adults there is less apparent distention, but measurement reveals an appreciable increase on the left side. The pressure exerted upon the left lung leads to a diminution in respiratory expansion on that side. The cardiac impulse may be wholly invisible or there may be a diffuse pulsation in the third and fourth interspaces, the whole ventricle being pushed forward and upward against the chest-wall by the fluid below and behind. Downward pressure of a large effusion may cause a distinct epigastric bulging or tumor. Percussion reveals a marked increase of pericardial dulness, the area of dulness having a somewhat pyriform shape with the broad base toward the diaphragm and the apex above toward the great vessels. The dulness is particularly extensive transversely, and may reach far toward the right nipple and to the left to the lateral or even posterior portions of the thorax. Rotch has pointed out the importance of dulness in the fifth interspace of the right side as an indication of effusion. The area of dulness changes somewhat, and sometimes considerably, with change of position of the patient. A most important sign is the extension of the area of dulness beyond the position of the cardiac impulse, if this remain visible or palpable. On auscultation the friction-sound is usually found to have disappeared, though, as stated before, a friction at the base of the heart may persist in spite of large effusion. At the same time, as the effusion increases, the heart-sounds become more and more weak or muffled until they disappear entirely—first, in the region of the apex, and lastly, even at the pulmonic or aortic cartilages. This disappearance of the heart-sounds, taken in connection with the percussion signs, is highly significant. When the effusion is very large the pressure upon the left lung may be so great as to lead to considerable compression of its lower lobe, with impairment of resonance and bronchial or broncho-vesicular breathing.

SPECIAL FORMS OF PERICARDITIS.

ADHERENT PERICARDIUM (*Chronic Adhesive Pericarditis*).—The manner of formation of adhesions between the layers of the pericardium by agglutination and subsequent organization of proliferated connective-tissue cells has been described. This may occur in simple fibrinous pericarditis or after the absorption of the serous exudation. It may also, though more rarely, occur when purulent effusions have been absorbed, and there is reason to believe that some cases are of tuberculous nature. In these instances the layers of the pericardium are greatly thickened, and the adhesions are apt to be extensive. In simple plastic cases there are often only a few long bands, which in no way impede the action of the heart and lead to little if any change in its structure. When, however, the adhesions are firm and short, the constant restraint offered to the cardiac movements leads to hypertrophy of the organ, sometimes reaching enormous proportions. There is thus established a compensation by which the circulation is properly maintained; but finally myocardial degeneration and dilatation result and the force of the heart becomes inadequate.

The **symptoms** are in no wise distinctive. They result merely from the failure of cardiac power. As long as this remains good, there may be absolutely no symptoms, and instances in which extensive adhesions are found at autopsy in which no clinical manifestations had been present are frequently reported. When fatty degeneration and dilatation of the heart supervene, visceral congestions, cyanosis, and œdema manifest the failure of compensation. The pulse becomes weak and irregular, and occasionally the *pulsus paradoxus*, in which during inspiration the pulse-wave is weak or almost inperceptible, is observed. The latter is not, however, a characteristic sign, as it occurs in some cases of large pericardial effusion and in indurative mediastino-pericarditis. In one of my cases the clinical features manifested were those of angina pectoris of most decided character, with gradually increasing failure of cardiac power. Not rarely adherent pericarditis leads to sudden death, especially on great exertion.

The **physical signs** are more significant. On inspection of the precordial region the heart's action is noticed to be jogging and irregular, and there may be seen with each systole a dimpling or retraction, instead of impulse, in the region of the apex. This retraction is most marked when there are adhesions between the pericardium and the pleura and chest-wall, and in some cases affects a considerable area. Its presence is not, however, absolutely diagnostic of adherent pericardium; and on the other hand it may be absent in marked cases. When the heart relaxes in diastole the chest-walls, retracted by the systolic traction, suddenly rebound, and a sharp diastolic shock is frequently noted. The same explanation has been offered by Friedreich for another physical sign, the "diastolic collapse" of the jugular veins, the rapid rebound of the chest-walls furnishing proper conditions for a sudden emptying of the veins. Percussion shows the heart to be enlarged, but the area of dulness has not the pyriform shape of pericardial effusion, and is not changeable with change of position of the patient. When there are associated pleuro-pericardial adhesions, the lung does not advance and restrict the cardiac dulness on deep inspiration; and the apex-beat may be firmly fixed in one position. The auscultatory signs may be wholly normal, or there may be a certain degree of muffling of the sounds if the pericardial layers are greatly thickened. A loud metallic character of the heart-sounds is noted when diaphragmatic adhesions draw the stomach upward against the heart. Intracardial murmurs are developed in cases in which dilatation of the cavities stretches the valvular orifices. The most common is a systolic mitral from insufficiency at this orifice. If the pericardial adhesions are long there may be a distinct and persistent friction-sound.

Pleuro-pericarditis (*Mediastino-pericarditis; External Pericarditis*).—In any case of ordinary or internal pericarditis the inflammation may extend through to the neighboring structures, but more commonly external pericarditis results from inflammation of the pleura or of the mediastinum. It is particularly marked in tubercular pleurisy and may be an important complication of malignant disease of the mediastinum. In acute cases there is the same plastic exudation seen in pleurisy and pericarditis or there may be collection of purulent effusion. In the later stages pleuro-pericardial adhesions and cicatricial bands extending in various directions constitute the morbid changes.

The **symptoms** vary largely with the exact position of the lesion. When there is simply inflammation of the pericardial layers with adjacent pleurisy the symptoms resemble those of pericarditis with excess of the ordinary manifestations. Dyspnœa in particular is apt to be urgent and the pain becomes more diffuse. The physical signs likewise are much the same, but to the ordinary pericardial friction, heard when the breath is held, there is added a pleural element, which may even overshadow the pericardial rub, when the patient makes full inspiration. Even when this double character is not detected it may be noted that the friction-sound is audible over a much greater area than is customary in pericarditis, and the point of greatest distinctness is apt to be found at the borders of the heart. In cases of cicatricial mediastino-pericarditis Kussmaul detected the *pulsus paradoxus* before referred to, explaining its occurrence by the assumption that at each inspiratory effort the bands of adhesions kink or press upon the aorta at its root. The same explanation is offered for the distention of the veins of the neck during inspiration. Neither of these signs, however, is to be regarded as absolutely distinctive, though their diagnostic value is considerable, when taken in connection with other signs.

Tuberculous Pericarditis.—Primary tuberculosis of the pericardium may occur in association with the same process affecting the other serous cavities, as a general primary tuberculosis of the serous membranes. There may also be a primary tuberculosis of the pericardium alone; and there is evidence to indicate that some of the cases of adherent pericardium discovered *post-*

mortem are really of tubercular origin. In most cases the pericardium is affected secondarily by extension of tuberculosis of the lymphatic glands, the lungs, or pleuræ, or miliary tuberculosis may affect the pericardium in common with other structures. The lesions may be those of simple plastic pericarditis without tubercles, but such a condition is rare. Usually the tubercles are found imbedded in the membrane, and are obscured from view by the fibrinous exudation on its surface. Serous, hæmorrhagic, and purulent exudations not infrequently result, and may be very large. Hæmorrhagic fluid in particular is highly suggestive of tuberculosis. The purulent exudation is sometimes thin, at other times quite thick and creamy. In late stages the pericardial layers are greatly thickened, covered with fibrinous or cheesy deposit, or united by fibrous adhesions.

The **symptoms** are never distinctive. A gradual onset, with irregular fever and increasing general weakness, and the discovery of hæmorrhagic effusion by exploratory tapping, would be almost positive indications. This evidence would lead to a certain diagnosis, if tuberculosis of the lungs or pleura were established at the same time. The detection of tubercle bacilli in the effusion is very difficult, though it has been accomplished in a few cases.

CANCEROUS PERICARDITIS.—Carcinomatous or sarcomatous involvement of the pericardium is a very rare condition, and is almost never primary. Usually there has been direct extension from malignant disease of the œsophagus or stomach, the mediastinal lymph-glands, the lungs, or the heart-wall, or metastasis from distant regions. The pericardium, besides the malignant new-growths, usually presents the evidences of simple inflammation with fluid exudation. Very frequently the fluid is hæmorrhagic.

Symptoms pointing to disease of the pericardium may be entirely absent, or there may be the clinical picture of ordinary pericarditis. Pain is sometimes sharp and lancinating in character; and the discovery of enlargements of the superficial lymph-glands may further create the suspicion of malignant disease.

Diagnosis.—It is highly important to recognize the occurrence of pericarditis in the course of rheumatism or the other diseases with which it is associated. Particularly in rheumatism the frequency of pericardial and endocardial complications makes careful daily examination of the heart imperative. A slight increase of fever, irregularity and rapidity of the pulse, or precordial distress may give the first warning; but the unequivocal sign is the characteristic friction-sound. Friction-fremitus is much less constant and distinctive. The friction-rub is recognized by its time and quality and the apparent nearness to the ear. It is distinguished from pleural friction by the disappearance when the breath is held, though in pleuro-pericarditis this distinction does not always hold. In such cases the greater diffusion of the sound and the added respiratory rhythm during inspiration may serve to make the diagnosis. The murmur of acute endocarditis may rarely be simulated when the pericardial rub is soft and single. The distinction is easily made by noting the greater softness and deep-seated character and the exact synchronism with the heart-sounds of the murmur in endocarditis, and the variability with pressure of the stethoscope and full inspiration in pericarditis. The difficulty of distinguishing a double aortic murmur from a basal friction-rub is more pronounced, but in this case also the general characteristics of the friction-rub are important distinguishing features; and the transmission of the valvular murmurs, the systolic upward into the vessels of the neck, the diastolic down the sternum, with the history of long-standing cardiac disease and the existence of cardiac hypertrophy, leave little doubt as to the diagnosis. Exocardial friction-murmurs, due to circumscribed pleurisy adjacent to the heart, acquire the cardiac rhythm and closely simulate pericardial friction. They are located on the outer border of the sac, and, although presenting cardiac rhythm, they are much affected by respiration and the associated symptoms suggest a pleuritic origin.

Pericarditis with effusion is sometimes difficult to distinguish from dilatation of the heart. In one case in my experience the diagnosis remained uncertain for several days, and later twenty-nine ounces of serous fluid were removed by aspiration. The development of dilatation is usually much less rapid, though instances of acute dilatation are occasionally met with. The area of dulness in dilatation very rarely has the pyriform shape seen in pericardial effusion, and, according to Rotch, dulness is not so frequently found in the fifth interspace of the right side. The presence of the cardiac impulse in the second, third, or fourth interspace, with extension of dulness further outward to the left, is highly significant of pericarditis, as is also the muffling or disappearance of the heart-sounds, especially when it occurs in succession from below upward. The cardiac impulse in dilatation is usually wavy and diffuse. It is rarely so weak as to be invisible or impalpable, and when such is the case the pulse is correspondingly weak; in pericardial effusion a comparatively strong pulse may be found when impulse has entirely disappeared.

Tumor of the anterior mediastinum may in some cases be difficult to distinguish, but the area of dulness and the character and position of the apex-beat give important indications, together with the more gradual development of the condition, and at times the presence of secondary growths elsewhere, as in the glands of the neck. Inflammation and abscess of the anterior mediastinum might also be mistaken for pericarditis. In these cases, however, the history discloses a traumatic cause and absence of rheumatism or the other precursory diseases of pericarditis. Fever and pain are more decided, and the area of dulness is irregular in outline. Finally, mediastinal abscess may point and break externally. In cicatricial mediastinitis the *pulsus paradoxus* has been considered an important sign.

As mentioned in the discussion of the symptoms, cerebral manifestations may be so prominent as to obscure the pericardial disease. Such cases usually occur in connection with acute rheumatism, and careful examination of the heart would reveal the existence of pericarditis. The presence of pericardial effusion may also be overlooked or obscured by a massive pleuritic effusion of the left side. In such cases it is at times impossible to arrive at a positive diagnosis, though careful physical examination will usually render a presumptive diagnosis justifiable. In one instance a circumscribed empyema between the inner aspect of the left lung and the pericardial sac produced a group of physical signs so strongly suggesting pericardial effusion that when I aspirated it was under the impression that the latter condition existed.

Prognosis depends largely upon the primary cause. The duration of the disease varies from two to several weeks, or it may be lingering and become chronic. In the idiopathic form, and in cases occurring in connection with rheumatism or infectious fevers, prognosis is highly favorable; in those with Bright's disease and pyæmic diseases it is most unfavorable. In any case the existence of purulent exudation and extensive fibrinous deposit makes the chance of recovery uncertain, and the danger of subsequent adhesive pericarditis in case of recovery very great. Extensive pericardial adhesions lead to progressive loss of cardiac power, and not rarely sudden death. In pericardial effusion death is usually slow, but sudden death from syncope may result after exertion. When pericarditis is complicated by endocardial lesions, by extensive pleurisy, or pneumonia, prognosis is correspondingly more grave; and when hyperpyrexia and urgent cerebral symptoms occur in rheumatic pericarditis, the outlook becomes gloomy. Tuberculous cases usually end fatally, but the possibility of termination in adhesive pericarditis cannot be overlooked; malignant cases are always fatal.

Treatment.—From the very outset it is imperative that the patient be placed in bed, absolutely at rest physically and mentally; and even in the mildest cases this rule is not to be departed from in the slightest. The effect of excitement and cardiac overaction in increasing the pericardial inflammation has been demonstrated beyond doubt; and in later stages, when effusion is present, there may be danger of fatal syncope if the patient be allowed to make undue exertion. The diet should be light, but nourishing, and as far as possible entirely liquid.

The important indications for treatment are, in the first stage, to limit inflammation and quiet the heart; in the later stages, to cause absorption of the exudation and to avert cardiac failure. Pain when severe will call for special treatment, and fever may demand attention.

Local applications to the precordium exercise positive influence upon the inflammatory process, and I am convinced that the early and repeated use of blisters affects profoundly the course of the disease. Milder counter-irritants, as by tincture of iodine, have fallen into disuse, and probably rarely possess any real value. In robust subjects the application of a few leeches or the use of cups is of great value, but they should be employed only in those in whom the general systemic power is good. When pain is a prominent symptom, the use of heat or of cold has been the customary treatment. Perhaps no local application exercises the same influence upon pain and the inflammation as does cold. An ice-bag or Leiter's tubes, or simply cold compresses, may be used, and under their action the heart will usually grow more quiet, the pain less severe, and doubtless the degree of inflammation is to some extent controlled. The internal use of mercury in small doses, preferably in the form of calomel, is useful, especially if exudation threatens to be abundant. It may be guarded with small doses of opium if necessary, since purgation should be avoided.

The following combination is often very acceptable :

℞. Pulv. digitalis,	
Mass. hydrargyri,	*āā* gr. x ;
Pulv. opii,	gr. v ;
Quininæ sulph.,	gr. xxx.

Ft. mass et div. in pill. No. xx.
Sig. One pill three or four times daily.

To quiet the action of the heart, aconite and veratrum viride are only occasionally useful. As a rule, it is far better to steady the heart in its action by the use of small doses of digitalis, but when cold is employed it may be unnecessary to use further treatment for this purpose. In cases where cardiac excitement is brought about by pain, or in any case when pain is present, small doses of morphine, especially by hypodermic injection, or Dover's powder act more happily.

When effusion declares itself, more vigorous means become necessary to promote absorption. The iodide of potash under these circumstances has great value, and particularly is this the case where effusion tends to become chronic ; but care must be exercised that the dose of iodide is not excessive. It is necessary to avoid derangement of the stomach, so that the dose should be small to begin with, to be increased from day to day as tolerance is shown. If 10 grains three times daily can be taken, it will usually be as effective as, and more safe than, larger doses.

At the same time, remedies directed to the action of the bowels and kidneys are desirable. Occasional doses of cathartics, such as Epsom or Rochelle salts in strong solution, or compound powder of jalap, may be used if the strength of the patient permit. For diuresis, digitalis, either as the tincture or in infusion, with vegetable salts of potash, forms a happy combination. If the effusion prove lingering, these remedies must be continued, and at the same time repeated blisters may be applied over the heart. Not rarely the cardiac and general bodily power is so much depressed that active stimulation by brandy or whiskey becomes a necessity, and in such cases also digitalis and strychnine prove valuable adjuncts in the treatment.

If the persistent use of these means fail to cause absorption of the fluid, or if, with large effusion, pronounced cardiac symptoms, such as irregularity and failing force of the pulse, cyanosis, increasing dyspnœa, and the like make their appearance, the immediate removal of fluid becomes necessary.

Paracentesis of the pericardium was first recommended by Riolan in 1649, but was not actually performed until 1840, though several unsuccessful attempts had previously been made. The point of election at which to insert the trocar is the fifth left interspace, about two or two and a half inches from the sternum ; roughly, therefore, in the region of the normal apex-beat. In nearly all cases the heart has been pushed up above this point, and lies in little danger of being injured. An ordinary trocar and canula of small size, with an aspirator, serves very well, but to avoid any chance of wounding the heart the trocar should be withdrawn into the canula as soon as the sac has been penetrated. Various special instruments have been devised, but can hardly be considered necessary in practice. A number of instances have been recorded in which unintentional puncture of the ventricle has occurred, but in few has much harm resulted, while in a number the relief afforded to intracardial distention by removal of blood has seemed of distinct benefit. The relief to the symptoms induced by paracentesis is usually decided. In the first case in which I performed the operation, in 1877, the patient, a young girl of seventeen years, was almost moribund when the pericardium was tapped yet the beneficial effect was most marked, and in a month the girl was well. At times the abstraction of a small quantity may give a start to the process of absorption, and the rest may disappear under medical treatment. So, too, when pleural effusion coexists, removal of the latter will often hasten resorption of the pericardial fluid. In cases in which effusion returns repeated tapping must be performed. Simple aspiration does not, however, suffice when the fluid is purulent. In these cases incision with establishment of free drainage is most important, but I have always felt that the irritation kept up by the friction of a drainage-tube against the heart was apt to prolong the inflammation. If the incision be large, drainage is readily established without a tube, and in my cases where a tube has been introduced I have regretted it.

Special indications at times arise calling for treatment. In rheumatic cases the continuous use of the vegetable potash salts throughout the case is often advisable, but the salicylates are without value, and may prove absolutely deleterious by their depressant action on the heart. Fever, though rarely urgent, occasionally becomes excessive, and is best controlled by hydrotherapic measures—either cool sponging or cold compresses. When hyperpyrexia is present, the immediate reduction of the temperature by cold packs or ice placed about the patient, or even by the use of the powerful antipyretics, antipyrin or phenacetin, gives the only chance of saving life. In the same cases violent nervous symptoms, such as delirium or sleeplessness, may call for the use of morphine in small doses. Chloral is less safe from its action on the heart, and is best used by enema.

The treatment of the special forms of pericarditis does not, in general, vary from that laid down. In adherent pericardium the cardiac power must be conserved by every possible care to avoid over-exertion or cardiac excitement, and by the continuous use of digitalis and strychnine or nux vomica in small doses.

During convalescence from acute pericarditis great care must be had to avoid fatigue or undue exertion, and the general strength may be restored by nourishing diet, iron, and other tonics.

HYDROPERICARDIUM.

Hydropericardium, or dropsy of the pericardium, is the condition in which the pericardial sac contains a fluid transudate of non-inflammatory nature. At nearly every autopsy a certain amount of clear, light-colored fluid is found in the pericardium. This probably exudes from the blood-vessels during the death-agony or soon after death, and is not to be considered as hydropericardium unless the quantity exceeds 20 or 30 ccm.

Hydropericardium may occur in association with the generalized dropsies of Bright's disease and of valvular heart disease, more rarely in emphysema of the lungs or hepatic cirrhosis. In such cases dropsy of the serous cavities is apt to coexist, especially hydrothorax. Extreme anæmia and cachexia may lead to hydropericardium by the hydræmic condition of the blood, and local causes, such as the pressure of an aortic aneurism or of mediastinal tumors, sometimes lead to stasis in the pericardial veins and serous transudation.

The fluid in hydropericardium is light yellow in color and quite watery; less commonly it may be slightly turbid or tinged by admixture of blood or biliary coloring matter. Occasionally the fluid is distinctly milky or chylous—chylo-pericardium. The quantity of fluid is not as great as in pericarditis with effusion, and the enormous dropsies formerly recorded were probably in reality inflammatory.

At post-mortem examinations the pericardium shows no indication of inflammation, though it is not rarely dull and opaque from interstitial œdema. The fluid may become somewhat consolidated in cases which have lasted some length of time, and transparent, jelly-like material may be found, especially posteriorly at the entrance of the cavæ; but fibrinous exudation invariably indicates actual pericarditis.

The **symptoms** are due to pressure, and are in every way similar to those of effusion in pericarditis. Sometimes transudation occurs so gradually that the symptoms are very slightly marked. The physical signs are likewise identical with those of effusion, excepting that the friction-rub is at no time present.

Diagnosis.—It is important to distinguish dropsy of the pericardium from inflammatory effusion. This, as a rule, causes no difficulty when the history of the case, the existence of dropsy in other portions, the absence of fever and of friction-sound are carefully considered. It is to be remembered, however, that in the late stages of pericarditis with effusion the failing heart-power may occasion general anarsarca. The remarks as to the diagnosis of the exudative stage of pericarditis should be consulted in the separation of hydropericardium from certain other conditions which may simulate it.

The **prognosis** of hydropericardium depends entirely upon the cause, but, on the whole, it must be regarded as a complication of grave import, and one which is only apt to occur in the last stages of the disease to which it is due.

The **treatment** must first of all be directed to the cause. The use of purgative and diuretic remedies would naturally suggest itself, and in cases in which the excessive quantity of the fluid directly endangers life aspiration of the fluid will prove of temporary benefit.

HÆMOPERICARDIUM.

Hæmopericardium, or blood in the pericardium, is a rare condition. It may result from rupture of an aneurism of the first part of the aorta, the coronary arteries, or the heart itself; from rupture of the heart-wall in myocarditis, or from penetrating wounds of the chest-wall, injuring small vessels or the heart itself. It may also occur in severe crushing of the thorax. Pericarditis with sanguineous effusion, such as occurs in cancerous and tuberculous pericarditis, or in the pericarditis of the aged or cachectic, cannot, strictly speaking, be called hæmopericardium.

The **symptoms,** when the amount of hæmorrhage is large, are those of rapid heart failure and of hæmorrhage in general. There is a sense of weight or compression of the heart; the pulse becomes extremely weak and irregular, the respirations shallow, and the patient is rapidly prostrated. Such are the symptoms when aneurisms rupture into the pericardium. In spontaneous rupture of the heart and in penetrating wounds of the pericardium the hæmorrhage occurs more gradually, and signs of heart failure are less decided, and may be wholly obscured by nausea or vomiting, with other indications of gastric or abdominal derangements.

The **physical signs** are those of liquid in the pericardium, but the quantity of blood rarely becomes as great as in pericarditis or hydropericardium.

Prognosis is most unfavorable, though in cases where only small arteries have been ruptured and little loss of blood has taken place gradual absorption and recovery may occur.

Treatment.—The patient must be placed absolutely at rest, and restorative remedies administered to support the heart. Paracentesis has been of value in a few cases.

PNEUMO-PERICARDIUM.

Pneumo-pericardium, or air in the pericardial sac, is a very rare condition, and quickly gives rise to secondary inflammation of the pericardium, with effusion of serous, or more frequently purulent, exudation.

Pneumo-pericardium may result from penetrating wounds of the chest or from perforation of an air-containing cavity within the body into the pericardial sac. Thus it may occur in ulcer or cancer of the œsophagus or stomach, from perforation of a cavity within the lung, or of pneumothorax. Graves has recorded a rare case in which an abscess of the liver communicated with the stomach and pericardium, and Stokes, one of rupture of the œsophagus in the attempt of a juggler to swallow a sword. The older writers, particularly

Laennec, believed that air might accumulate in the pericardial sac much as do serous and other effusions, during life, in the death-agony, or immediately after death, and that gases might be produced by decomposition of a pericardial exudate.

In a few cases the air in the pericardial sac has been absorbed without any indication of pericarditis having been present, but generally inflammation and exudation, particularly purulent, speedily supervene, the membrane then presenting the characteristic appearances of pericarditis.

The **symptoms** are those of oppression of the heart, and subsequently fever and the evidences of pericardial effusion.

The **physical signs** are more distinctive. There may be bulging of the precordial region and on percussion a tympanitic note is detected. Later, when effusion is present, a movable area of dulness with overlying tympany is very characteristic. The auscultatory phenomena are often remarkable. At first, when air alone is present, the heart-sounds may be distant and feeble, but have a decidedly metallic quality. Later a friction-sound is noted, and when fluid and air are present the coexistence of splashing or churning sounds, with friction or new-leather sound, and the heart-sounds, all of peculiar metallic timbre, constitutes an association of signs hardly to be mistaken. At times the sounds are so loud as to be heard several feet away from the chest, and in the remarkable case of Stokes the sleep of the patient and his wife was disturbed by the loudness of the sounds.

Diagnosis.—A dilated stomach may give rise to a metallic quality of the heart-sounds, but is readily distinguished by the outline of the tympanitic area and by careful physical examination of the heart. A sacculated pyopneumothorax or a pulmonary cavity situated anteriorly on the left side may also cause confusion, but either is recognized by the discovery of the normal cardiac outline and by respiratory sounds associated.

Prognosis is not necessarily grave in traumatic cases, but where the underlying cause is a perforation of cancer or an ulcer of some neighboring structures, the outlook is entirely hopeless.

The **treatment** must be directed to supporting the heart by stimulating remedies, and to limiting as far as possible the subsequent pericarditis. Opium will frequently be needed for its quieting effect upon the patient, and an ice-bag applied over the heart is often of service. When distention is great, paracentesis may be performed, and in cases in which purulent fluid is determined free incision might be practised if the underlying cause permitted the hope of ultimate recovery.

DISEASES OF THE ENDOCARDIUM.

BY WILLIAM PEPPER.

ACUTE ENDOCARDITIS.

INFLAMMATION of the lining membrane of the heart generally affects the valves, though the endocardium of the cavities and the chordæ tendineæ may also be involved. It is customary to distinguish two forms of acute endocarditis, a simple and a malignant, but in reality there is so gradual a transition of one form into the other, and the causes are so frequently identical, that the classification must be regarded as of a clinical rather than of a strictly scientific nature. At most we can say that malignant endocarditis is but a severe type in its anatomical and clinical aspects, and not a disease distinct from simple endocarditis.

SIMPLE ENDOCARDITIS.

SYNONYMS.—Verrucose, Warty, Exudative Endocarditis.

Etiology.—Acute endocarditis is very rarely a primary and independent affection, and in the cases where no antecedent disease is discoverable the suspicion of a latent or deep-seated infective or rheumatic disease must be entertained.

The most important cause by far is acute articular rheumatism, from 60 to 85 per cent. of all cases having this disease as the origin of the endocarditis, and from 30 to 50 per cent. of all cases of rheumatism presenting endocarditis as a complication. It is in the severer cases especially, and in those in which a large number of joints are affected, that the cardiac disease is most apt to supervene, and in point of time it will be found that the onset usually occurs in the first or second week of the rheumatic attack, so that rheumatic endocarditis seems rather a special localization of the rheumatic disease than a secondary affection to which a prolonged and debilitating disease has rendered the system liable. It is essential to note also that endocarditis may occur as a complication of non-articular rheumatic fever, either alone or associated with tonsillitis or myalgia or wry-neck. It is in these cases particularly that the primary cause of the disease is apt to be overlooked. Chorea evidently has a certain causal relation with endocarditis in early life, especially in the cases in which a distinct rheumatic history is obtained. The existence of a systolic murmur has, however, been too frequently accepted as positive evidence of the existence of endocarditis. In many such it is probable that the murmur has been purely functional; yet the frequency of actual endocarditis in chorea is unquestionably great, and few fatal cases fail to show some warty excrescences on the valves.

Endocarditis may occur as a complication in almost all of the infectious diseases. It is common in scarlet fever, less so in measles, diphtheria, erysipelas, small-pox, and typhoid fever, and in all of them it is a late complication, occurring very frequently during convalescence. The frequency of an endocardial complication in phthisis and, especially, in pneumonia has been until recently overlooked, though both the simple and the ulcerative forms occur in a notable proportion of the cases. Gonorrhœa and various septic and pyæmic diseases more frequently give origin to malignant endocarditis.

Besides these diseases which more or less directly cause endocarditis, there are a number of conditions in which the general state of the system predisposes to it. Among these Bright's disease is the most prominent, though here endocarditis is less frequent than pericarditis. Cancer, diabetes, gout, and peliosis rheumatica must also be reckoned as occasional predisposing causes, acting by the general wasting or debility consequent upon them.

The possibility of extension of inflammation in myocarditis or pericarditis to the endocardium may be admitted, but such an occurrence is very rare.

Age exercises a distinct predisposing influence, altogether aside from the fact that certain of the diseases enumerated above are more frequent in early life, for it has been found that rheumatism is more apt to occasion endocarditis in children than in adults. Added to this, the frequency of infectious fevers and chorea during childhood makes the disease one of early life rather than of maturity. The influence of sex in itself is less important than has been maintained, greater frequency of chorea and rheumatism in girls explaining their apparently greater susceptibility. In adults the disease is more frequent in men, in whom rheumatism is more common than in women. Pregnancy and the puerperal state unquestionably furnish a strong predisposition, especially when previous endocarditis had been present; and the existence of chronic valvular disease predisposes to acute attacks.

The question of the direct exciting cause has not as yet been definitely settled. In the diathetic diseases, such as Bright's disease, the presence of poisonous substances in the blood may be important, but the tendency of late is more and more to ascribe to bacteria the rôle of exciting agents. No specific micro-organism has, however, been detected; on the contrary, a number of bacteria have been found, some of them the specific organisms of diseases to which the endocarditis was secondary, while others are forms not yet elsewhere detected. The pneumococcus, the staphylococcus pyogenes aureus and albus, the streptococcus pyogenes, the bacillus pyogenes fœtidus, the bacillus of typhoid fever, and several micrococci and bacilli found only in this disease, are the forms which have been isolated. One or more of these may be found in a single case. The bacillus of tuberculosis, which has been detected in a few instances, is regarded by Ziegler as of accidental occurrence; a deposit from the circulating blood after the endocarditis has been established. It is not, however, possible to find bacteria of any kind in every case, nor is it certain that they are infective agents in all cases in which they are found, though their position in the tissues and the nature of the tissue-changes surrounding them almost certainly establish their pathogenicity.

From certain experiments at artificial production of the disease in animals it appears that slight injury of the endocardium is a necessary antecedent condition, and this explains very well the frequency of the disease on the left side of the heart in post-natal and on the right side in fœtal endocarditis, the blood-pressure being greater under each condition in the cavity in which the disease occurs. It also explains the greater frequency of mitral than aortic disease, as the pressure and consequent liability to injury are much more pronounced, according to Sibson, in the former.

Morbid Anatomy.—The changes which take place affect primarily the endothelial lining, the subendothelial connective tissue, and the blood-vessels. The earliest visible change is a moderate opacity or cloudiness of the endothelial surface, the result of necrotic changes in the cells. Subsequently the small blood-vessels become engorged, and round-cell infiltration in the subendothelial tissues is apparent on microscopical examination. At the same time, cellular proliferation occurs and formative cells are numerous in the later stages. As a result of these processes there is formed a localized thickening of the valve, which, with the roughening due to the degeneration of the lining cells, invites the deposition of fibrin from the circulating blood. In this way the small bead- or cap-like deposits of fibrinous material which have given the name of verrucose or warty endocarditis become established, and not, as was at one time urged, by exudation from the underlying vessels. When the disease is fully developed the appearances vary to a large extent with the locality and with the intensity of the inflammation. There may usually be seen along the line of contact of the valves—that is, along a line a little removed from the free edge and where the maximum contact occurs on closure—a row of small bead-like elevations, somewhat irregular in shape and sometimes of distinct cauliflower appearance. Later, with additional deposit of fibrin, masses the size of a split pea may be formed, but this size is attained less frequently in simple than in ulcerative endocarditis, and less commonly on the aortic than the mitral valve. The mitral valve is by far the most common seat; the aortic is next in frequency; while right-sided endocarditis rarely occurs except during fœtal life. Of 300 cases reported by Sperling of post-natal endocarditis, the left heart was affected alone in 268, the right alone in 3, and the two sides coincidently in 29. Among these cases the mitral valve was affected 255 times; the aortic, 129; the mitral alone, 157 times; and the aortic alone, but 40 times. On the other hand, in studying fœtal endocarditis Rauchfuss found 192 cases in which the right alone was affected, against 15 involving the left side. Such cases as that of Bland-Sutton, in which warty elevations were discovered at autopsy of an eight-months' fœtus, leave no doubt as to the identity of the fœtal and post-natal disease. With the valves the endocardium of the heart-walls may be affected, or more commonly the chordæ tendineæ; but affection of these parts alone is rare, though certain observers have insisted upon the frequency of the left auricle as a point of origin. The disease is transferred from one point to others by contact and attrition, and it has been maintained that it may be primarily induced by friction of an old and calcareous lesion of a valve with neighboring parts.

Underlying the inflammatory lesion of the parietal endocardium there is always a certain amount of myocarditis from extension, and in violent cases the disease may involve the whole thickness of the wall. Pericarditis or implication of the right heart may thus be brought about by extension, though such a condition is very rare.

In cases which run a favorable course resolution occurs by gradual absorption of the exudate and of the surface deposit of fibrin, but complete resolution is rare. It is vitally important, however, to insist on the possibility of its occurrence even in marked cases, so that it may constitute the aim of our treatment. More commonly the subendothelial cellular infiltration and formative cells pursue their natural tendency to organize, and localized thickening of the valve results. In marked cases the whole valve is thickened and stiff, and later suffers the ordinary changes seen in sclerotic endocarditis. Neighboring leaflets may be bound together, or the valve may be firmly attached to the wall of the heart or aorta. When the chordæ tendineæ are affected, the weakening may lead to rupture during the acute stage, or subsequently they become thickened and rigid. It is precisely this tendency to chronic sclerotic changes with valvular defect which gives to acute endocarditis its greatest danger.

The fibrinous deposits on the valves are naturally liable to break off and be carried as emboli throughout the body. In case the left heart is affected, the kidneys, the spleen, the brain, and the skin are the localities in which the emboli most frequently become lodged; when endocarditis involves the right heart embolism of the lungs is frequently observed. As a consequence of the embolism, hæmorrhagic infarctions of the organs named may result, or if micro-organisms be present abscesses may be produced. The latter, however, are rare, and in general embolic manifestations are less common in simple endocarditis than in the malignant form or in acute attacks supervening upon chronic valvulitis.

Symptomatology.—The variable and insidious character of the onset and course of acute simple endocarditis cannot be too strongly insisted upon. Some cases run through their entire course, to the establishment of chronic valvular disease or to a fatal result, in which no definite symptoms have called attention to the endocardial complication. Generally, however, it will be noted in the course of rheumatism or of any other febrile disease in which endocarditis develops, that the fever increases, the pulse gains in frequency, and the patient suffers dyspnœa with præcordial distress. None of these symptoms may be decided, and, especially in severe rheumatic fever, they may readily be overlooked. As, however, the tendency to endocarditis in acute infectious diseases is now well known, repeated physical examinations must be made as a matter of routine, and the occurrence of any unusual disturbance of pulse or temperature should at once awaken strong suspicion. When endocarditis occurs at the onset of a case, the temperature may rise abruptly, with the usual symptoms of fever, flushed face, coated tongue, thirst, and restlessness, and not infrequently excessive sweating and sudamina make their appearance. Thus, I have not very rarely observed, in children of rheumatic diathesis, an abrupt onset of fever up to 104° or even to 105.5° F., with acute endocarditis and tonsillitis, but with no articular symptoms whatever. The pulse at first is quick, but fairly strong; soon it grows more feeble, and in grave cases dichrotous. Extreme cardiac weakness is nearly always an indication of coexisting myocarditis. In such cases the cardiac impulse, at first decided, becomes more and more feeble until it finally becomes entirely impalpable, the pulse at the same time growing weak and irregular, and the lips and finger-tips or the whole surface of the body more or less cyanotic. The sense of præcordial distress before referred to usually suggests a weight on the heart or the constriction of a band, but sometimes severe pain is experienced in the præcordial region or in the abdomen, and it may occasionally have decided anginal characters. In young children the pain is very apt to be referred to the epigastrium —a circumstance which increases the danger of mistakes in diagnosis. The accompanying dyspnœa is moderate, though in the rare cases in which destructive changes have occurred in the valves or embolic manifestations in the lungs the most extreme grades of dyspnœa and orthopnœa may be met with. Nervous symptoms, such as continued sleeplessness, delirium, stupor, and coma, are sometimes noted, but are usually the result of complications or of the underlying rheumatic disease rather than of the endocarditis itself.

Finally, the occurrence of embolism may give to the disease an entirely different picture. The commonest seat of embolism is in the kidneys, after which the spleen, the brain, and the skin follow in succession, and in right-sided endocarditis the lungs are the first to be involved. A deep-seated lumbar pain coming on suddenly and followed by albuminuria or hæmaturia would almost certainly indicate renal embolism; as would sharp pain in the left hypochondriac region and swelling of the spleen, embolism of this organ. Embolism of the cerebral vessels is fortunately very rare, but is recognized by various palsies, according to the location of the embolus, and by the sudden disturbance of consciousness. A petechial or purpuric eruption on the skin and on the various internal surfaces manifests the lodgment of small emboli in these portions, and retinal embolism with impairment of vision or complete blindness is occasionally observed. Sharp colicky pain in the abdomen, the result of embolism of the mesenteric arteries, is a rare but urgent symptom. These embolic symptoms are much oftener noted in ulcerative or malignant than in simple endocarditis.

Physical Signs.—As a rule, the physical signs are much more distinctive than the symptoms, though they also are subject to great variations, and may be simulated in conditions other than endocarditis. Inspection and palpation confirm the subjective sensations by revealing increased rapidity and force of the cardiac impulse. The patient lies on the back or inclines to the left side, and in the late stages, when the cardiac power fails, distention of the cervical veins and pallid cyanosis may become decided. Palpation sometimes discovers an endocardial thrill. By percussion the area of præcordial dulness at first is found but slightly affected, and such may be the case to the end of the disease. When, however, myocarditis is present and dilatation results, or when the valvular lesion becomes subacute and compensatory hypertrophy is established, the area of dulness increases, and at the same time palpation discovers in the one case diminution, in the other increase, of the impulse. The most important sign, however, is the bellows murmur. This is a soft and blowing systolic sound, best heard at the apex and imperfectly transmitted to the left. It is present in over half of all cases of rheumatic endocarditis, but it must always be remembered that murmurs in every way similar to it may occur in the course of rheumatism and the infectious fevers when endocarditis is not present. The bellows murmur of endocarditis is the result of mitral leakage or of roughening of the endocardium of the ventricle. In the former case increase of tension of the pulmonary circulation, with accentuation of the second sound at the pulmonary cartilage, results, and still later tricuspid regurgitation makes its appearance in a notable percentage of cases. Preceding the development of the murmur, it is generally noted that the first heart-sound is prolonged or harsh, and sometimes it is a double sound. Murmurs at the other valves and diastolic murmurs are much less common, excepting that at the tricuspid valve already mentioned. In children a double murmur at the mitral orifice is not unusual, and was present in nearly one-fourth of the cases of Cheadle's series. Systolic murmurs at the aortic cartilage may be anæmic, though more frequently organic; those at the pulmonary orifice are almost always anæmic. A diastolic aortic murmur is occasionally detected, and, according to Sibson, results from inflammatory softening and relaxation of the aortic valves with failure of the Arantian bodies to completely close the orifice.

Diagnosis.—Attention has already been called to the great risk of overlooking the existence of endocarditis in acute febrile attacks without marked localizing symptoms. Nothing but an unvarying rule of making thorough physical exploration a part of the examination of every acute case will obviate grave oversights. We are at least equally apt to fail to notice the development of endocarditis in the course of infectious diseases, and this is pre-eminently true of acute rheumatism. Indeed, this can only be accomplished with certainty when the heart is carefully examined at every visit to the patient. In many cases beginning rapidity or irregularity of the pulse, præcordial distress, or dyspnœa may be noted, but the frequency of an insidious onset without symptoms makes the daily examination of the heart imperative.

When, however, a murmur has been discovered by auscultation, there still remains the possibility that it may be the result of febrile dilatation or anæmia, and not of endocarditis. The distinction is to be made by the character of the sound and its distribution, and by the changes in the heart which follow. Anæmic murmurs are usually very soft, and are best heard over the base of the heart; they bear no relation to cardiac enlargement, though the causes which produce the anæmia may also lead to cardiac dilatation, and in anæmia there is no evidence of obstruction to the circulation. The murmur of myocarditis and dilatation, however, is frequently heard at the apex, and there is some enlargement of the heart, but the accentuation of the second sound at the pulmonary cartilage is rarely present, and tricuspid regurgitation almost never. When a murmur is detected at the first examination of a rheumatic case, the question may arise whether the endocarditis be old or recent. In such cases the harshness of the sound and extensive alteration in the size of the heart would be almost certain indications, and the diagnosis would receive still greater certainty by the discovery of sclerotic arteries at the wrist or temple.

The distinction of simple from malignant endocarditis in many cases offers the greatest difficulty, and may only be possible when the progress of the case toward recovery or a fatal termination has become evident. Ordinarily, however, the history of an antecedent septic or pyæmic disease, the existence of highly irregular fever, with chills or profound nervous symptoms, the presence of albumin and tube-casts in the urine, and the evidence of destructive lesions of the valves with multiple embolism are sufficient to indicate the malignant nature of the case. It must be remembered, however, that there are on the one hand cases of ulcerative endocarditis of milder type, and on the other hand instances of simple endocarditis approaching closely to the type detailed.

The diagnosis of pericarditis has been sufficiently considered in the discussion of that disease.

Complications and Sequels.—The most important direct complication is myocarditis, and it is to this that the untoward symptoms of the later stages are attributable. Very rarely the inflammation may extend to the pericardium, but usually, when endocarditis and pericarditis coexist, they are both dependent upon a common cause, and not the one upon the other. Pleurisy and pneumonia are dangerous complications, resulting, as a rule, from failing pulmonary circulation, and therefore manifesting themselves in the dependent parts of the lungs. Rupture of one of the valvular segments of the chordæ tendineæ or of a papillary muscle is probably commoner than would appear, and is denoted by sudden onset of extreme cardiac failure, and often by development of a very loud whirring murmur. Such an accident, however, is more apt to occur in malignant endocarditis. The embolic complications have been described under the Symptomatology.

The most common sequel of simple endocarditis is chronic valvular disease with stenosis or insufficiency at the orifices. It is probable also that some cases beginning as simple become malignant endocarditis, but of this there can, of course, be no direct proof. It is not unusual to find in the endocardium whitish patches of sclerosis, some of which doubtless followed acute endocarditis; and when the underlying myocardium is simultaneously involved, a cardiac aneurism may result.

Prognosis.—Uncomplicated cases of simple endocarditis rarely prove fatal;

but when extensive myocarditis or pericarditis coexists, or when a severe primary disease has induced great depression, endocarditis must always be looked upon as a dangerous condition. The tendency to relapsing endocarditis is sometimes marked in rheumatic cases, and in such the constant reappearance of inflammatory lesions so prolongs the case and depresses vitality that the outlook for final recovery becomes a gloomy one, and the establishment of chronic valvular disease, in case of recovery, almost certain. In all cases the liability to organic valvular changes is very great. The chances of complete recovery with the restoration of the normal condition of the valve is much greater in children than in adults.

Embolic manifestations, gastro-intestinal disturbances, pneumonia, and other complications necessarily increase the liability to fatal termination.

The duration of the disease is always uncertain. Sometimes the symptoms disappear and convalescence is established in the course of two or three weeks; in others a fatal termination, or the decline of fever and active symptoms with establishment of valvular disease and hypertrophy, is delayed until several months have elapsed. Recurring endocarditis is especially apt to occur in rheumatic cases.

Treatment.—The prophylaxis of endocarditis is important. It concerns the avoidance of the disease in subjects predisposed to it and in those suffering with the febrible diseases, and especially rheumatism, in the course of which the heart is likely to become involved.

If a child be known to have the rheumatic diathesis, close attention is to be paid to every detail of hygiene; and while a tonic and hardening regimen is cautiously adopted, care is to be used to avoid violent exercises which might overtax the heart, render it specially vulnerable, and thus favor its implication in any acute attack.

In acute rheumatism the danger of cardiac complication must never be lost sight of. It is in the first place essential that absolute rest of body and mind shall be maintained. Sibson's statistics confirm the result of our observation that no other influence does so much to obviate endocarditis as complete rest associated with a careful avoidance of draughts and sudden changes of temperature.

The value of salicylates for this purpose is more than doubtful, though their use may lessen the frequency of endocarditis by shorting the duration of the rheumatic disease or by relieving pain and thereby diminishing cardiac excitement. I am in the habit of applying a small blister to the præcordia in cases of acute rheumatism when irritability of the heart exists, although no sign of endocarditis may as yet be detected.

If, unfortunately, the heart becomes involved, a continuance of absolute rest is even more essential. Visitors should be excluded from the sick-room; talking must be prohibited; nothing which will excite the heart can be permitted. The diet should be light and nourishing. The temperature of the room should be kept uniformly at from 72° to 75° F. I am convinced of the value of small blisters applied over the præcordia at successive points and at intervals of three to five days. A series of blisters of one or one and a half inches in diameter are greatly to be preferred to one or two large ones. In violent cases leeches may be applied to the region of the heart, but I have rarely resorted to them.

If pain be complained of, an ice-bag or Leiter's tubes applied to the præcordial region will be found to afford decided relief; or in cases in which the pain is more severe it may be necessary to use small doses of opium or morphine, for the cardiac excitement resulting from pain serves to intensify the endocardial inflammation.

The salicylates rarely seem to exert any beneficial influence, and care is needed to avoid depression of the heart or derangement of digestion if they are used long or freely. Rarely I have met cases where these remedies alone gave relief to præcordial distress in rheumatic endocarditis accompanied with myocarditis.

Potassium iodide may be given in carefully graded doses, and later may be associated with minute doses of mercury in the hope of preventing further exudation. Whether these or any other internal remedies can secure absorption of the exudation and fibrinous deposit already formed is problematical. It is well to use them, if given in such small doses as not to impair digestion. It seems, however, that the lapse of time with prolonged scrupulous hygienic care and continued mild counter-irritation are the potent agents for this purpose; and, as already said, they may effect complete restoration in young subjects.

The excitement of the heart's action usually calls for cardiac sedatives in the early stage of acute endocarditis. Aconite or veratrum viride are the most effective and safe; but it must be remembered that there is often a natural tendency to cardiac depression in the late stage, and therefore such remedies are to be used with caution. When the force of the impulse is weak and the circulation is manifestly failing digitalis will be found to steady the action of the heart and to increase its power. At the same time use may be made of such general stimulants as alcohol and strychnine.

Fever does not usually call for active medication. The temperature may rise to a considerable height, but rarely maintains it for more than a few hours at a time. Quinine will usually be found to exercise a controlling influence, if it does not actually reduce the temperature. It may be given in doses of three to five grains by the mouth, or, when the stomach is irritable, in suppositories of four to eight grains each; and in the latter case the extract of opium and digitalis may be added if indicated by pain or cardiac weakness. Such a combination as the following may then be used:

℞. Quininæ sulphatis,	ʒj vel ʒiss;
Extracti opii,	gr. iv;
Pulveris digitalis,	gr. vj vel gr. xij;
Olei theobromæ,	q. s.—M.

Fiant suppositoria no. xii.

Sig. One suppository every three or four hours.

During convalescence the rigid enforcement of hygienic measures and careful regulation of diet and exercise must be insisted upon ; and iron or other tonics will prove of value in hastening the restoration of the strength of the patient.

MALIGNANT ENDOCARDITIS.

SYNONYMS.—Ulcerative, septic, mycotic, diphtheritic endocarditis. The name malignant seems preferable to the more commonly used one, ulcerative, and to the others in that it indicates no constant form of lesion and no theory of causation, but merely the well-defined clinical tendency to an unfavorable termination. To each of the other terms it may be objected that, while appropriate in some cases, it is inexact in others. Malignant endocarditis was first described by Kirkes in 1851, and afterward more thoroughly studied by Charcot and Vulpian in France and Virchow in Germany.

Etiology.—The existence of primary malignant endocarditis cannot be denied, though it is rare. Cases may arise in persons in perfect health or those under treatment for chronic valvular disease, in which none of the diseases which usually precede the endocardial complication is discoverable.

In the great majority of cases malignant endocarditis is secondary, following rheumatism, various infectious diseases, or septic processes of traumatic or puerperal origin. The frequency of rheumatism as a cause has certainly been overestimated. This may be in part due to confusion of the simple with the malignant form, and in part to the fact that the pain in the joints may be called rheumatism, although in reality only a symptom of the infectious processes with which the endocarditis is associated. When malignant endocarditis does develop in connection with rheumatism, it usually appears early in its course. On the other hand, the painful affection of the joints which may appear after the endocarditis is established is frequently septic in nature, though it may be rheumatic.

Of the infectious diseases with which malignant endocarditis is associated, the most important by far is pneumonia, 11 of 100 fatal cases of this disease studied by Osler having presented this endocardial complication. It may also arise in the course of scarlet fever, diphtheria, small-pox, typhoid fever, tuberculosis, or dysentery, but in all of these diseases it is rare. Attention has recently been directed more particularly to gonorrhœa as a cause of endocarditis of both simple and malignant type.

When malignant endocarditis results, as is not uncommon, from a septic process either puerperal or traumatic, the diagnosis is usually clear. The uterus or its appendages present the characteristic lesions of membranous or diphtheritic inflammation, phlebitis, and the like; or the external injury in traumatic cases shows more or less advanced suppuration. Occasionally the endocardial disease arises when traumatic lesions have been unaccompanied by any evidence of infection. Puerperal endocarditis generally occurs within a few days or a week after labor, but may be delayed until the woman has left her bed. It is more apt to follow abortions than labor at term, and in a few cases has occurred during pregnancy and led to abortion or miscarriage. Of other septic processes, which occasion malignant endocarditis, phlegmonous inflammations, necrosis of bone, and periostial disease are prominent examples.

I have known the disease to arise after exposure to the damp, unhealthy air of a low-lying office that had been overflowed in a freshet; and, in another case, to the damp, foul air in a deep trench dug in the city streets.

Old valvular lesions predispose strongly to malignant endocarditis. Goodhart found sclerotic valves in 61 of 69 cases, and Osler in more than three-fourths of his series. Age exercises a less potent though still distinct predisposing influence. The majority of the cases occur between the thirtieth and fortieth year, but Cheadle records one in a child of eight years, and instances in young people as well as in old persons are frequently reported. No special influence can be attributed to sex; but exposure, malhygiene, and excessive use of alcoholic drink render the system liable to this as to other infections. Chorea is rarely associated with this form of endocarditis.

The micro-organisms which have been found in malignant endocarditis are the same as those mentioned in the etiology of simple endocarditis. The pyogenic micrococci are especially frequent, and in few cases does careful investigation fail to discover some form.

Morbid Anatomy.—The lesions vary widely in different cases, being either vegetative, ulcerative, or suppurative. The vegetations of the malignant form are generally much more pronounced than in the simple form, though otherwise similar. They consist of irregular, often cauliflower-like, masses of granular fibrin, entangling desquamated endothelial cells, round cells, and micro-organisms. In some cases by detaching the vegetation the base is found to be distinctly ulcerated; in other cases it is only slightly roughened and elevated. The vegetations themselves may be quite tough; more commonly they are friable and easily detached.

In the cases to which the term ulcerative is especially appropriate, and which form a large proportion of the whole number, distinct ulcers are found upon the valves or mural endocardium, sometimes shallow, more commonly deep and destructive. Occasionally the ulcer is so covered by fibrinous deposit as to escape detection until the vegetations are removed, and in a few cases spots of superficial erosion or roughening may be found before the actual ulceration has occurred. In cases of suppurative type small purulent collections are seen between the valvular reduplications or at the base of the valves and involving the walls of the cavities. These small abscesses may remain intact and be seen at autopsy; or they may break and thus produce superficial ulcers.

The situation of the lesions is practically the same as in simple endocarditis, the mitral and aortic valves being affected much oftener than the tricuspid and pulmonary. In the 204 cases collected by Osler the mitral valve was alone affected in 77, the aortic in 53, the tricuspid in 5, and the pulmonary in 4. The involvement of the right heart is most frequently found in cases due to traumatic and puerperal pyæmia. Coincidently with disease of the valves or independently the mural endocardium is involved in a considerable number of cases, the points of greatest frequency being the upper part of the septum ventriculorum and the posterior wall of the left auricle. Very frequently areas adjoining the ulcerations or vegetations are secondarily affected by contact during the cardiac movements, and in rare instances the lesions may spread to the lining of the aorta.

The local results of the ulcerative lesions are mainly of a destructive nature. The valves may be so eroded that they are reduced to mere stumps; the leaflets or the septum may be perforated; one side only of a leaflet may be destroyed and an acute valvular aneurism result; or, in case of involvement of the mural endocardium, even partial cardiac aneurism may occasionally occur. The formation of valvular or cardiac aneurism is, however, a rare occurrence, and still less commonly aortic aneurisms have been observed at the seat of endarterial ulcers. Purulent myocarditis or even pericarditis may result from direct extension, but the former is more often due to miliary embolism of the coronary arteries, and the latter generally arises coincidently with endocarditis, rather than secondarily.

The remote lesions of malignant endocarditis are the outcome of septic intoxication or of embolism. The spleen is usually enlarged as in infectious diseases, and the parenchyma of the kidneys and liver suffers the degenerative changes of fever. To these lesions may be added those of embolism. In case of mitral or aortic disease this involves the systemic circulation, and the emboli are lodged in the spleen, the kidneys, the brain, the cutaneous vessels, or the retina; in case of tricuspid or pulmonary valvulitis the lungs are the seat of the embolic lesions. In many cases simple hæmorrhagic infarcts result, but in the more decidedly pyæmic cases, those especially which result from traumatic or puerperal processes, suppurative infarctions or multiple miliary abscesses are observed; and sometimes a central point of suppuration is surrounded by a more extensive zone of hæmorrhage. In the skin, the serous surfaces, and the retina minute hæmorrhages may result from embolism or from degenerative changes in the vessels unassociated with emboli. Cerebral embolism leads to lesions of the meninges or of the deeper structures. Meningitis is especially common in the cases occurring in the course of pneumonia, as, indeed, it may be met with in this disease altogether apart from endocarditis. There may further be meningeal or deeper hæmorrhages, or embolic softening, simple or suppurative in nature. In some instances multiple pyæmic deposits have been found in the brain.

Pleurisy, parotitis, and embolism with secondary ulcerations in the stomach and intestines are among the rarer lesions. A moderate degree of local peritonitis may be present when the spleen is the seat of infarctions.

Symptomatology.—The clinical course of malignant endocardititis presents such variety in different cases that no description will closely apply to all of them. In nearly all instances, however, there is an underlying, if not conspicuous, pyæmic element, which gives to the disease its most marked peculiarities. The onset is usually abrupt and marked by a decided chill. If the disease arises in the course of a febrile affection, considerable exacerbation of the temperature occurs; or if the patient had been previously healthy, pyrexia is at once established. During the continuance of the disease irregular remittent or sometimes definitely intermittent fever is maintained, the evening temperatures being 103° to 104° in decided cases, and the morning a few degrees lower or even subnormal. With this irregular fever repeated rigors and drenching sweats may be associated, and may add to the depression of the general strength of the patient, which is in any case a marked symptom. Local indications may or may not be present. Generally there is slight oppression or the feeling of constriction at the heart; more rarely there is actual pain, but severe pain is very unusual. The cardiac action is excited, the pulse is rapid, and often irregular and weak. A slight subjective sense of palpitation may be the only symptom directing attention to the heart. Dyspnœa is less decided than the rapidity of the pulse would lead one to expect, unless pulmonary lesions complicate the case.

The tongue is coated, and in many cases becomes brown and dry. Sordes collect about the teeth. The stomach is frequently irritable in the early stages, and vomiting may prove an urgent symptom. The abdomen is often distended with gas, and diarrhœa may alternate with constipation, or may be so persistent and severe as to resemble cholera. The spleen is found enlarged and tender. The urine nearly always contains albumin, and may even be tinged with blood. Tube-casts, epithelial or granular, are frequently found.

Nervous symptoms are rarely absent. They may merely consist of headache, restlessness, or slight muttering at night, but in severe cases, and especially toward the close, active delirium and a soporose or completely stuporous condition may be developed; and when cerebral embolism has supervened the occurrence of various palsies may serve to indicate the nature of the cerebral complication.

The whole appearance of the disease may be altered by the supervention of embolic manifestations. Sharp pain or tenderness and swelling of the spleen denote infarction and localized peritonitis about this organ. Small emboli of the kidney may not declare themselves by any definite symptoms. In the case of larger infarcts, however, pain in the lumbar region and hæmaturia are distinctive symptoms. Albuminuria alone occurs in nearly all cases, and is due to parenchymatous degeneration of the kidney, but when emboli obstruct the renal vessels the quantity of albumin increases notably. The manifestations of cerebral embolism depend upon the seat and the resulting lesions. Consciousness is nearly always lost, the patient becoming stuporous or comatose, and palsies result from occlusion of vessels supplying the motor cortex. Dimness of vision or extensive inflammation of the eye may follow retinal embolism, and colicky pain, gastric disturbances, and diarrhœa, sometimes hæmorrhagic in nature, give evidence of the occlusion of the gastric or intestinal vessels. Jaundice is an occasional symptom, and may be due to the septic and febrile degeneration of the liver or to embolism of one of the branches of the hepatic artery, as has been seen in a few instances. The skin may also present a vivid red erythematous rash, especially in the early stages, and later petechiæ are not uncommon. The latter may be due to minute emboli or to degeneration of the vessels without occlusion. They are most numerous upon the abdomen and chest, and sometimes have a distinctly papular as well as petechial character.

Varieties.—The great diversity of the symptoms requires the description of certain types of the affection which were recognized by Kirkes and described in his earliest writings.

In a large number of cases the disease closely resembles typhoid fever in its symptomatic indications, and it is customary to speak of this as the typhoid form. In certain cases, in which the onset is not abrupt, but gradual and attended with malaise or other prodromal symptoms, the resemblance to typhoid fever at its onset may be most deceptive. Later, nervous and abdominal symptoms become pronounced, the patient sinking into a delirious or stuporous condition, picking at the bed-clothes, and slipping down to the foot of the bed as in typhoid fever. The tongue becomes dry and brown, sordes collect around the teeth and gums, the abdomen is distended, the spleen enlarged, the bowels are apt to be relaxed, and tenderness in the right iliac fossa may be present from the first. To this may be added the eruption, which, though not like the characteristic rash of typhoid fever, is simulated in certain cases of the latter disease. The temperature is high and usually remittent, but may at times be continuous at 103° or 104° during the entire course of the disease.

In another group of cases the disease is manifestly of pyæmic nature, and to such the term "arterial pyæmia," applied by Wilkes, is not inappropriate. It is this type that occurs most frequently during puerperal septicæmia or after necrosis of bone or external suppurations. The onset is abrupt and attended by a decided chill. Throughout the disease the temperature is highly remittent or even intermittent, and attended by more or less periodical chills and sweats. The patient becomes prostrated, the tongue is brown, the breath offensive, the skin is sallow or jaundiced, and emaciation may proceed rapidly to an extreme degree. Embolic processes of a suppurative type and a petechial eruption may finally call attention to the cardiac lesion.

Sometimes in the course of chronic valvular disease, apparently without cause, fever is suddenly developed and evidences of acute endocarditis become more or less pronounced. In some cases there is nothing but a moderate acceleration of the pulse and respirations and the occurrence of irregular fever to indicate the endocardial disease; in others pronounced symptoms of the typhoid or pyæmic type may mark the case.

In a young man recently under my care for chronic aortic disease, ulcerative endocarditis arose, apparently without cause, and for over four months no more decided symptoms occurred than slight increase in pulse-rate and dyspnœa and moderate elevation of the temperature. Finally splenic infarction supervened, and death soon followed. Similar instances have been recorded where the duration was over a year.

Special mention must be made of the cases where, with irregular fever of remittent type, copious sweats, and distinct endocarditis, articular symptoms appear. Various joints may be affected in a more or less fugitive manner, or one or two of the larger joints may be distinctly inflamed. It may be difficult for some time to decide that the fever is not rheumatic and the endocarditis and arthritis dependent upon it.

Physical Signs.—The malignant, like the simple, form of endocarditis may present few signs in certain cases. The most frequent is the systolic blowing murmur heard in the region of the apex, and generally indicative of disease of the mitral valve. It must be remembered, however, that systolic murmurs, not at all dependent on endocarditis, are often heard during the course of fevers. An aortic systolic, or more rarely diastolic, murmur may be present, and the latter especially is significant of valvular disease. In all cases it will be found that the sounds alter from day to day in character and position of maximum intensity. Inspection and palpation merely reveal the overaction of the heart, and in protracted cases percussion may show a slight enlargement, but in ordinary cases this is rarely found. The existence of previous chronic valvular disease would of course be indicated by the altered character of the physical signs, in accordance with the particular lesion present.

Complications.—The most important complications are those which result from embolism, but they have been sufficiently detailed. Pericarditis and myocarditis from extension or from embolism are conditions of great gravity, and manifest themselves by the unusual and early depression of the heart's action and by physical signs varying with the exact nature of the lesions. Pneumonia and pleurisy are occasionally met with, though the former is more frequently antecedent to the endocardial disease. The pleural effusion is not rarely purulent in nature. Gastric and intestinal derangements may sometimes reach a severe grade, even without embolism, and vomiting and excessive diarrhœa, almost choleraic at times, may continue throughout the case. Meningitis is most commonly met with in the malignant endocarditis following pneumonia. It may be an early complication, and its symptoms may completely dominate the case.

Diagnosis.—The diagnosis of malignant endocarditis is often extremely difficult from the fact that the cardiac condition may be latent or overshadowed by severe complications. The disease is most frequently mistaken for typhoid fever, rheumatic fever, or ordinary pyæmia. The distinction from typhoid fever is especially difficult when the endocarditis is obscure in origin or primary and when prodromal malaise is noted. As a rule, however, the onset of malignant endocarditis is abrupt, the temperature is irregular, there are more or less well-defined chills and sweats, and there may be great rapidity of the pulse, oppression, or pain to direct attention to the heart. The abdominal distention and splenic enlargement of typhoid fever are usually more decided than in malignant endocarditis, and the eruptions are essentially different in typical cases. The frequency with which precedent organic cardiac disease will be discovered is an important point, as is also the much greater frequency of albuminuria with casts in malignant endocarditis.

Rheumatic fever may be closely simulated for a time when the case presents no clear septic origin and is marked by high, irregular fever, copious sweats, and arthritis. The endocarditis would be naturally expected, but the splenic enlargement, the embolic symptoms, the petechiæ, the albuminuria with casts, and the total failure of antirheumatic remedies will soon establish the diagnosis.

It is hardly proper to speak of the diagnosis from pyæmia, since malignant endocarditis is in reality a pyæmic disease. It is important, however, to discover the source of the pyæmic manifestations, and this may offer no little difficulty when the cardiac symptoms are not pronounced. In the absence of pain or other subjective cardiac symptoms, the rapidity of the pulse and the embolic manifestations would probably draw attention to the endocardial lesion, and physical examination would confirm the suspicion of malignant endocarditis. In cases of the pyæmic type the regularity of the paroxysms of chill, fever, and sweat may be such as to simulate very closely quotidian or tertian malarial fever, but examination of the blood and the general condition of the patient readily exclude this disease.

Difficulty may arise in certain cases occurring in the course of chronic valvular disease to distinguish the malignant from the simple form of endocarditis; but even in the mildest cases the temperature is more irregular and protracted, embolism and albuminuria with casts are more frequent, and the prostration of the patient more marked in the former than in the latter.

When the complications, such as meningitis, excessive diarrhœa, or pulmonary infarction and abscess mask the cardiac condition, careful physical examination alone will prevent error in diagnosis.

Prognosis.—It is difficult to determine whether the disease ever terminates otherwise than fatally. Cases of the protracted form in the course of chronic valvular disease have been known to recover, but it is doubtful if these belonged to the class of simple or of malignant endocarditis. Decided cases are always fatal. The duration varies from a few days to many months. The most rapidly fatal are those in which a decidedly malignant pyæmic condition has preceded the endocardial complication.

Treatment.—The management of malignant endocarditis is practically that which obtains in other forms of pyæmia, with attention to special indications. The diet of the patient should be nutritious, but easily assimilated, and if vomiting interferes with the taking of food, rectal alimentation must be instituted at once. Whiskey or brandy and quinine are doubtless the most suitable remedies to combat the septic intoxication and to maintain the powers of the patient. In addition to these, it may necessary to still further stimulate the heart by means of strychnine and digitalis, but it is best to avoid the latter until the evidence of increasing cardiac weakness and irregularity of the pulse are marked. In the earlier stages the over-action of the heart may call for treatment which will steady its action. Cold applied to the præcordial region, either by a bladder filled with cracked ice or by Leiter's coils, will usually accomplish the purpose, and is preferable to cardiac depressants.

CHRONIC ENDOCARDITIS; VALVULAR DISEASE.

SYNONYMS.—Sclerotic endocarditis; Interstitial endocarditis. Chronic endocarditis and chronic valvular disease are practically synonyms, since it is the endocardium of the valves which is affected in the great majority of cases.

Etiology.—Two classes of cases may be distinguished—those which follow acute endocarditis, and those which are from the beginning chronic in nature.

Cases of the first class may, in most instances, be traced to an attack of acute rheumatism, though the endocarditis of chorea and the infectious fevers forms the starting-point in a certain number. The frequency with which articular rheumatism is complicated by acute endocarditis has been referred to in the description of the latter disease, and also the regularity with which the acute form of endocarditis passes into a chronic condition. In general it may be stated that of all cases of valvular disease over one-half may be traced to a rheumatic origin. It must, moreover, be recalled that vague and aberrant types of rheumatic disease are sometimes the occasion of valvular affections, and there is reason to think that a slow and primarily chronic endocarditis may occur in persons who have never had any acute manifestations of rheumatism beyond certain indefinite pains. Chronic valvular endocarditis of rheumatic origin is more common in youthful persons than in those beyond middle age, and in a great majority of cases it affects the mitral valves.

All must have noted the frequency with which cases of unsuspected chronic valvular disease are met in children or young adults. Often the patient can refer to no acute attack connected with the heart. It is evident that in far more cases than is commonly suspected acute endocarditis occurs insidiously as an expression of rheumatism, as a complication of scarlatina, rubeola, or diphtheria, or as an idiopathic affection.

The second class, or that in which the endocarditis is chronic from the first, results from a variety of causes, for the most part the same as those which induce arterio-sclerosis. Among these causes syphilis, alcoholism, and gout are prominent, and in each the existence of noxious substances in the blood seems to play an important part in the occurrence of endocarditis. Excessive muscular labor is an important cause which has not been fully appreciated by most writers. The constant high tension of the arterial system and the strain upon the valves doubtless occasion slight injuries to the endocardium, which afterward set on foot the chronic sclerotic process (Roy and Adami). Bright's disease and arterio-sclerosis are closely allied to muscular strain in the manner in which they lead to valvular disease, but they may themselves be the result of the same causes as those producing the valvular disease. Chronic malaria, various cachexiæ, and chlorosis have been considered among the causes, but the evidence is less certain in these conditions. In a small number of cases the valvular disease seems traceable to pregnancy and the puerperal state.

The popular belief in an hereditary predisposition to valvular disease seems to have some foundation in fact, though perhaps in many cases the tendency is to be explained by the liability of certain persons to rheumatism and other conditions which lead to valvular affections. Some years ago a family was under my observation in which the father, mother, and two sons were subjects of chronic valvular disease. The father and one son had been under treatment for acute rheumatism, while the other son and mother were said to have been affected. Three of the cases have since died, each of them suddenly; the fourth is still living. Virchow pointed out that there is a special predisposition in cases of hypoplasia of the heart and aorta, such as occurs in some instances of chlorosis; and the frequency with which malformed valves are found to be thickened and indurated is too striking to be considered a coincidence. In each of the seventeen cases of bicuspid aortic valves reported by Osler the leaflets were sclerotic, and Lloyd found the same in the aortic valves of a child of but thirteen months.

The age at which valvular disease is most apt to occur differs with the valves considered. Fœtal endocarditis affects the right side of the heart in an overwhelming majority of cases. After birth the greater number of cases arise during adolescence and early adult life, when rheumatism and infectious diseases are common; and in this case the mitral valve is generally involved. Aortic disease is much more common in persons of advanced years, but may be met with in young men, especially those whose occupation involves unusual muscular exertion, as in the case of blacksmiths, miners, and soldiers during campaigns. In the case of soldiers it has been held that the frequency of syphilis is the important etiological factor, but, as Myers pointed out, syphilis is quite as common among sailors, and valvular affections much less so.

In itself, sex probably exerts no special predisposition, but, on the whole, women are more frequently affected than men. The explanation of this is perhaps to be found in the greater frequency of rheumatism and chorea among young women and girls. Mitral stenosis, in particular, is common in women, being nearly four times as frequent in women as in men in the statistics of F. J. Smith. Aortic disease is about three times as common in men as in women, according to the same observer, and here the frequency of gout and syphilis and muscular strain offers a ready explanation.

A small number of cases is directly traceable to traumatism. The sudden and severe strain accompanying a fall or crush is supposed by Peacock to be the immediate cause in these cases, and various forms of valvular laceration have been recorded. In examining the heart soon after the patient had been subjected to a heavy fall or crush, I have in at least half a dozen cases detected a decided systolic murmur, apparently of mitral origin, where I know by earlier examination that the heart had previously been healthy. In most cases this has passed away gradually under the influence of prolonged rest, but it has persisted in at least two instances in the form of chronic valvular disease.

Morbid Anatomy.—Beginning in acute endocarditis or as a chronic process from the first, the earliest change in the valves is a moderate thickening along the line of contact; that is, in the case of semilunar valves on the ventricular face near the free edge and involving the Arantian body, and in case of the auriculo-ventricular valves on the auricular surface. The endocardium becomes dull and opaque, and the thickening is nodular or uniform. Subsequently the process extends to the other portions of the valves, so that they become uniformly thickened and lose their customary elasticity. The minute changes involved consist in proliferation of the endothelium and infiltration of the subendothelial connective tissue with round cells, and subsequent organization into connective tissue. In the later stages the sclerotic tissue undergoes contraction, and the valves in consequence become curled and distorted, and may be reduced to mere stumps. In other cases, and particularly after acute endocarditis, neighboring leaflets are agglutinated, at times so accurately that the original edges are scarcely visible, and together they suffer the further changes seen in separate segments. Similarly, though far less commonly, one or more of the leaflets may be firmly attached to the adjacent ventricular wall, or, in the case of the aortic valves, to the intima of the aorta. From the valves the sclerotic changes extend to the neighboring structures. When the aortic valves are involved, the tissues at the insertion of the leaflets become thickened, and a more or less dense and inelastic ring is formed, from which the distorted valves project into the vessel. In the case of the mitral valve the chordæ tendineæ become thickened and rigid, the process extending from the edge of the valve toward the papillary muscles, which themselves are finally invaded at their ends. The thickened chordæ contract and draw the valvular segments forward into the ventricle, giving rise to an appearance not inaptly likened to that of a funnel extending into the ventricle. At the same time, the valvular ring may be affected, as in the case of the aortic valves. With the union of the adjoined edges of the mitral leaflets the orifice may in this manner be reduced to a mere slit or buttonhole, or, more rarely, there may be a rounded orifice which will admit but the point of a finger or a quill. In other cases of mitral disease the contraction of the chordæ and the papillary muscles leads to a widening of the orifice, but the curling of the edges of the valves is less commonly observed than in the aortic leaflets. In the latter also a somewhat funnel-like appearance is sometimes produced by union of the cusps and contraction of the free edge, but this is much less frequently observed than in the case of the mitral valve.

The final changes in sclerotic valves are similar to those seen in patches of sclerosis in arteries. Fatty degeneration and necrotic changes lead to the formation of atheromatous ulcers, which may be seen unchanged or further altered by deposition of lime-salts. The most extreme degrees of valvular distortion may be thus produced; and sometimes the valves in their entirety, as well as the subvalvular ring, may be completely calcified. The formation of true cartilage has been observed in a few instances, but the terms "ossification" and "chondrification" of the valves, formerly in use, were applied from a misconception of the real nature of the process.

The thickened and ulcerated or calcareous valves invite the deposition of fibrin from the circulating blood, and may occasionally become capped with warty deposits not unlike those of acute endocarditis, or the latter disease, in either its simple or malignant form, may actually supervene. It is important to distinguish between the ulcers of malignant endocarditis occurring on sclerotic valves and the atheromatous ulcers above referred to; but little difficulty is experienced when the necrotic appearance and the tendency to extensive calcareous deposition found in the latter are carefully observed.

The alterations in the valves lead to derangement of their functions—on the one hand, to backward regurgitation of the blood, when the segments are so curled, distorted, or stiffened as to be incapable of accurately closing the orifice; and, on the other hand, to obstruction to the outward flow of the blood when the agglutination or rigidity of the segments or the infiltration and contraction of the valvular ring render the orifice narrow or constricted. It is customary to speak of the former as insufficiency of the valves, of the latter as stenosis or constriction.

The existence of either condition must necessarily lead to imperfect circulation of blood through the heart, and, unless some relief be offered, this stagnation will be followed by serious consequences; and indeed, in cases of sudden destruction of valvular segments by traumatism, a fatal issue is frequently observed. In chronic valvular disease, however, the imperfections in the valvular mechanism are developed slowly, and coincidently nature provides compensatory changes in the structure of the heart which serve to maintain the proper circulation. It has been shown by Jaksch that when one segment of the valve is shortened by sclerosis, a compensatory lengthening of the other segments may develop and prevent regurgitation; but cases of this kind are highly exceptional. The usual method of compensation is naturally by an increase in the size and power of the heart-walls. For example, it is clear that the effects of a narrowing at the aortic orifice may be overcome by increased force of the ventricle, whereby the pressure under which the blood is propelled makes up for the contraction of the orifice. So too in cases of regurgitation at the same valve, hypertrophy of the ventricle would provide for a sufficient expulsion of blood at each systole to maintain the arterial supply, and at the same time to allow for the quantity returning to the heart through the insufficient valve. In this way hypertrophy of the heart in valvular disease may be explained on purely mechanical principles, the amount of enlargement being proportioned to the necessities of the circulation. Coincidently with hypertrophy the cavities become more or less enlarged, from the fact that a greater quantity of blood is contained within them, and the pressure exerted in systole is higher, than in the normal heart. The degree of enlargement of the heart thus produced may reach extreme proportions. Frequently the weight is from 20 to 25 ounces; Fagge and Dulles each reported a case in which the weight was 48 ounces; and extreme weights have been recorded—as 53 ounces by Beverly Robinson, 57 ounces by Clark, and even 64 ounces by Stokes. The heart may remain in this condition for various periods of time, but eventually further changes are observed in its structure, and secondary alterations occur in remote organs and tissues.

As a result of advancing imperfections in the valves or of associated changes in the root of the aorta and coronary vessels, the circulation in these arteries is interfered with and cardiac degenerations supervene. The same result follows or is hastened by general systemic disease or increasing anæmia. The muscular structure suffers parenchymatous and fatty degeneration, which may be seen in scattered areas as dull, lead-colored, or yellowish spots or streaks, visible through the endocardium. Very rarely the muscular structure becomes uniformly fatty. In aortic lesions, and especially when the coronary arteries are sclerotic, fibroid changes may form an important element, and nodules of cicatricial myocarditis are seen within the muscular substance and invading the papillary muscles. The same lesions, however, may be due to direct extension from the endocardial lining in mural or even valvular endocarditis. Eventually fatty degeneration also occurs. The heart-substance, weakened by such degenerative changes, is no longer able to withstand the increased endocardial pressure, and excessive dilatation of the cavities results, in consequence of which the circulation cannot be maintained and venous congestion of distant organs ensues.

The remote effects of failing circulation fall most naturally and with greatest weight upon the lungs. Even when compensation is fairly maintained, there is excessive distention of the pulmonary vessels, and when unusual efforts tax the cardiac power such congestion increases in extent. With failing compensation increasing degrees of distention of the pulmonary vessels gradually lead to permanent dilatations, and even a high grade of varicosity may be seen. The lung-structure loses its elasticity, and is dark and œdematous. Gradually the interstitial tissues proliferate, and the condition known as brown induration is developed. The sluggish circulation sometimes leads to the formation of thrombi in the pulmonary vessels, and extensive infarctions or areas of atelectasis are produced. Pulmonary collapse may also result in a purely mechanical way from pressure of an enlarged left auricle upon the lung or bronchus, as in the cases of King and of Friedreich, or of a dilated and hypertrophied ventricle, as I have seen.

Congestion of the liver causes enlargement of the organ, and on section the characteristics of the nutmeg liver, in which the distended intralobular veins form a dark centre surrounded by the lighter-colored liver-cells, are observed. Later the liver-cells undergo fatty degeneration, and finally the whole organ may become reduced in size, indurated, and cyanotic. Congestion and catarrh of the bile-duct may be present at the same time and lead to important clinical manifestations. The kidneys are enlarged and deep-red in color ; later they become smaller and indurated ; and a genuine nephritis may arise as a complication. The spleen is enlarged and firmer than normal ; its substance is deeply cyanotic. The mucous membrane of the stomach and intestines is swollen and catarrhal, and there may be hæmorrhagic extravasations and subsequent erosion, forming superficial ulcers. When the congestion has been long continued the mucous membrane is thickened and the rugæ are prominent.

The roughened and ulcerated condition of the valves and the presence of fibrinous deposits upon them readily explain the frequency of embolism. Another source from which emboli are sometimes derived is the formation of thrombi formed within the cavities or in the peripheral veins, owing to the sluggish circulation of the blood. The nature and situation of the embolic manifestations are the same as in the case of acute endocarditis, the spleen, kidneys, and brain being the most frequent seats. More rarely emboli may occlude the vessels of the extremities, of the retina, and of the mesenteric arteries, or, in cases of disease of the valves of the right heart, the branches of the pulmonary artery. Emboli derived from venous thrombosis are also apt to lodge in the vessels of the lungs.

Chronic endocarditis affecting the mural endocardium is of much rarer occurrence than that seen upon the valves, but may arise independently or as a result of valvular disease where a roughened valve impinges upon the wall of the heart. The appearance presented by such lesions is usually that of a dense white spot slightly elevated above the surface. The underlying muscular substance is regularly involved in its superficial portions, or even extensive myocarditis may result. Stenosis of the conus arteriosus may be brought about by the subsequent contraction of an annular zone of sclerotic endocarditis and myocarditis, the right heart being most frequently so affected, and generally during fœtal life. Occasionally, however, such a lesion is found in the left heart, and *post-natal* in origin.

The special changes in the different portions of the heart will be most appropriately considered in the description of the individual valvular lesions.

MITRAL INSUFFICIENCY.

Mitral insufficiency is by far the commonest form of valvular defect, but there is difficulty in estimating the exact frequency, because, on the one hand, clinical evidence is uncertain in many cases, and, on the other hand, there is no invariable test by which moderate degrees of incompetence of the valve may be ascertained post-mortem when clinical signs point to its existence. Unassociated with other lesions, it probably constitutes from 30 to 40 per cent. of all cases, and in at least as many more it is associated with other valvular defects.

It is slightly more common in women than in men, and for the most part affects younger persons than do lesions of the aortic valve. In the great majority of cases mitral regurgitation may be traced to a rheumatic origin.

Insufficiency of the mitral leaflets may be due to the changes in the valves resulting from endocarditis, or may be "relative" when the normal segments are unable to close the orifice, owing to excessive dilatation of the ventricle or to imperfect apposition resulting from improper action of the papillary muscles. As a result of chronic endocarditis the leaflets may be curled or contracted, so as to be incompetent, but more commonly are retracted by thickened and stiffened chordæ tendinæ or by agglutination and rigidity of the segments themselves. There is very often a certain amount of stenosis associated.

Relative insufficiency, due to dilatation of the ventricle, is common in cases of idiopathic dilatation, or may follow aortic lesions with failure of the left ventricle. The result in either case is inability of the mitral segments to close the dilated orifice, and regurgitation, with all the secondary changes in the auricle, lungs, and right heart, speedily ensues. Compensating conditions are naturally less apt to develop, since the relative mitral insufficiency is itself a terminal development. In the course of fevers or of cardiac degenerations from other causes slight regurgitation may result from failure of the proper muscular tone necessary to the exact apposition of the valvular segments.

The primary effect of regurgitation of blood into the left auricle is dilatation of this cavity, and subsequently there is some hypertrophy of its muscular tissue to compensate for the increased demand required in its contraction. The amount of hypertrophy of which the auricles are capable is, however, never great, and may often be practically ignored. The increased tension in the auricle and the hypertrophy of its walls cause increased expulsion of blood at each auricular systole, and therefore overfilling and dilatation of the left ventricle, which in turn hypertrophies sufficiently to maintain the normal pressure in the aorta in spite of the regurgitation into the auricle. The most extreme degree of ventricular hypertrophy may be thus produced, and may persist for many years. The backward pressure of the blood at the same time leads to dilatation of the pulmonary vessels and to the pulmonary changes incident upon congestion of the lesser circulation, and finally hypertrophy of the right ventricle results from the increased pulmonic tension. By these successive changes the circulation through the heart and lungs is maintained at approximately a normal condition until the myocardial degenerations, which manifest themselves by failing compensation, have developed. At this stage the left ventricular cavity undergoes increased dilatation, and the right ventricle, unable to cope with the increasing regurgitation, becomes excessively dilated, permitting tricuspid regurgitation and general venous engorgement. The changes described may remain stationary for considerable periods of time. Thus, it is not unusual to find moderate mitral insufficiency, with hypertrophy of the left ventricle and but little enlargement of the right ventricle, persisting for years, and even when dilatation of the right ventricle has occurred from improper exertion or intercurrent pulmonary disease, restoration to the previous condition may often be secured by rest and suitable treatment. On the other hand, there is constant danger when the heart is in this state of delicate equipoise that strain or fatigue will overtax it and break the compensation, whereupon alarming symptoms may develop abruptly.

Physical Signs.—*Inspection.*—The apex-beat is displaced to the left and downward according to the degree of enlargement of the left ventricle. Sometimes, and especially in children, there may be decided bulging of the præcordial region and cardiac impulse is generally diffuse. When the right ventricle has become dilated, epigastric pulsation is usually observed, and I have even seen a distinct pulsating tumor in this region, no doubt the congested and enlarged left lobe of the liver pressed forward by the right ventricle. Occasionally no apex-beat can be detected, and the epigastric pulsation alone is visible, the enlarged right ventricle, from its position anteriorly to the left ventricle, displacing the latter from the chest-wall. These extreme cases of pulsating liver or of prominent pulsating epigastrium seem to require for their full development a state of relaxation, with or without degeneration, of the diaphragm. When compensation is failing, the lips, fingers, nose, and ears grow dusky and general cyanosis may be observed. Jaundice is occasionally present in slight degree.

Palpation.—The apex-beat is found displaced and is abnormally strong. There is often in addition diffuse pulsation, and occasionally a systolic thrill may be felt by placing the palm of the hand and the fingers flat upon the præcordium. The pulse is often entirely normal, though on exertion it becomes rapid and perhaps irregular. With failing compensation irregularity may become decided. The sphygmographic tracing is in no wise characteristic, showing at most irregularity.

Percussion.—The cardiac dulness is primarily increased toward the left, sometimes as far as the anterior axillary line. Some increase in an upward direction along the left border of the sternum may indicate the auricular distention, and the dulness of the right ventricle may extend beyond the right borders of the sternum and quite to the mammary line in extreme cases.

Auscultation.—The characteristic sign of regurgitation at the mitral orifice is a systolic murmur, best heard at the apex, and most strongly transmitted to the left axilla and even to the back. This murmur varies much in length, force, and quality. In contradistinction to hæmic murmurs the mitral systolic murmur is but seldom soft, and even more rarely is it musical. It may be so hard and harsh as to be audible at a distance of six or even twelve inches from the chest-wall, and to be transmitted not only to all parts of the chest, but along the spine down to the lumbar region and upward to the top of the head. It must not be supposed that the force and quality of the murmur are safe indications of the gravity of the valvular lesion: the contractile power of the ventricle counts for much in this respect, and a very serious lesion may be attended with but a weak murmur. The first sound is modified or partly concealed, or even entirely obliterated, by the murmur. The second sound at the apex may be weak, but at the pulmonic cartilage is nearly always sharply accentuated. When right ventricular dilatation becomes extreme, a systolic murmur of tricuspid insufficiency may be superadded, but is sometimes difficult to distinguish from the existing mitral murmur. Where the enlarged right ventricle has displaced the left ventricle from the chest-wall, I have known the mitral murmur to grow faint and almost inaudible, and to return to its previous character when compensation has been restored. I would call special attention to the cases where, owing to such enlargement of the right side of the heart, the impulse is largely produced by the right ventricle, and when a mitral systolic murmur may be inaudible at the apex-beat but become clearly marked at a little distance to the left.

It should be remembered that softer systolic murmurs, more or less localized at the apex, may be hæmic in origin or produced by the expulsion of air from the overlying lingula of the left lung. The sharp localization of such murmurs, and in the case of the latter its increase after full inspiration, serve to distinguish them from the murmur of mitral insufficiency.

Even when the murmur is organic it may be noted in some cases that it is affected by respiration, being considerably reinforced during respiration, or else heard best during held expiration; or it is influenced by the position of the patient, and heard much more clearly in the recumbent position; or it varies in intensity at different examinations, owing to varying degrees of cardiac activity, so that it may even be temporarily absent and be recalled by exertion.

Naunyn and later Balfour pointed out that in some cases of mitral regurgitation the murmur is heard with maximum intensity in the second or third interspace near the left border of the sternum, and that this is probably due to the superficial position of part of the left auricle at this point. In such cases confusion with pulmonic stenosis might arise, but the murmur has not the upward transmission of a pulmonary murmur and is usually at the same time audible over the apex.

MITRAL STENOSIS.

Mitral stenosis is far less common than regurgitation, but the conditions are often associated, and in a majority of cases of stenosis insufficiency coexists, though the narrowing is the dominating condition. It is more frequent in women than in men, the proportion being about four to one, and is generally met with in young persons. The evidence in favor of congenital stenosis is not so certain as has been held, though a few undoubted cases have been observed.

Rheumatism is the most common cause, and my observation agrees with that of some other observers, that it follows the subacute forms rather than the acute. Certainly mitral stenosis often develops insidiously, and cannot be traced to a definite time of origin. The morbid changes in the valve consist of thickening and rigidity of the segments and chordæ tendineæ, more or less fusion of the segments at their edges, and finally calcareous infiltration. The valve is thus converted into a rounded or flattened and rigid funnel projecting into the ventricle. In the former case the orifice is reduced to a narrowly contracted ring scarcely admitting the point of a finger; in the latter—and this is much more common—the orifice is slit- or chink-like, and commonly known as the "button-hole mitral." It is evident that with such high grades of rigidity there is inability to close as well as open, and regurgitation is therefore always present, though of slight degree. On the other hand, a certain amount of stenosis may develop in the course of insufficiency as the valvular changes become more marked. Stenosis may be produced at the mitral orifice when the valves are normal by infiltration of the valvular ring and by the presence of large calcareous masses projecting from the wall of the ventricle. Such cases, however, are extremely rare.

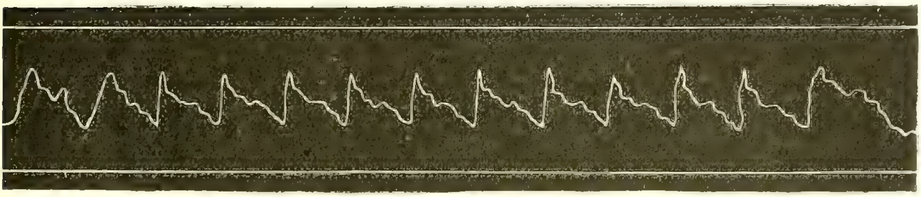
The cardiac changes following mitral stenosis are manifested in a backward direction. The obstacle offered to the passage of blood through the mitral orifice leads to dilatation of the left auricle, sometimes of extreme degree, as in the case of Friedreich referred to. Usually some hypertrophy accompanies the dilatation, and the auricular wall may be twice its normal thickness; but in the late stages the walls are very thin. The distention of the left auricle in turn causes congestion of the pulmonary vessels and secondary changes in the structure of the lungs. No other valvular lesion so constantly produces extreme grades of pulmonary congestion. In many cases a condition of hypertension is maintained in the lesser circulation for years, the lung-tissue in such cases slowly undergoing the sclerotic changes which lead to cyanotic induration, and the walls of the blood-vessels becoming at the same time more or less sclerosed. In a recent case I found decided atheroma of the pulmonary vessels to their remotest branches, with thrombosis in several, and extensive hæmorrhagic infarction, rendering the left lung almost completely solid and airless. Finally, hypertrophy and dilatation of the right ventricle supervene, and serve to maintain the proper circulation. With failing compensation the right ventricle becomes more and more dilated, the tricuspid valves are no longer able to close the dilated orifice, and general cyanosis presages the fatal termination. The left ventricle in cases of pure stenosis of the mitral orifice is little enlarged, and may even atrophy. Usually, however, when stenosis is not extreme and insufficiency coexists, hypertrophy of the left ventricle is observed, though rarely attaining great proportions. The absence of ventricular hypertrophy in mitral stenosis is readily explained by the fact that the left ventricle is constantly deprived of its normal supply of blood, and that the intraventricular tension is therefore always below the normal. The right ventricle is never capable of as decided and persistent hypertrophy as is the left, but cases of moderate stenosis at the mitral orifice may be well compensated for years.

Physical Signs.—*Inspection.*—The impulse of the heart may be seen in the normal position, and may be of ordinary strength. More commonly it is weaker, and sometimes disappears entirely. In cases of associated insufficiency and enlargement of the left ventricle the apex-beat is seen outside the nipple-line. The hypertrophied and dilated right ventricle very often causes prominence, and in case of children sometimes decided bulging and pulsation, of the lower sternal and epigastric regions. The congestive enlargement of the left lobe of the liver assists materially in such epigastric prominence. In rare cases a presystolic pulsation of the left auricle has been observed. The cervical vessels are often full, and sometimes pulsate at each ventricular systole. In these cases tricuspid regurgitation may generally be suspected. General cyanosis marks complete failure of the right heart.

Palpation.—The cardiac impulse is usually weak, and may wholly disappear when dilatation of the right ventricle reaches a high grade. Epigastric pulsation is often decided. An almost certain characteristic of mitral stenosis is the presystolic thrill. This is felt as a somewhat prolonged "cat's purr," ending in a sharp systolic shock. It is most distinct in the region of the apex, and grows in intensity up to the sharp impulse. It is not constantly present in mitral stenosis, and may appear and disappear in the same case from time to time. When present it is diagnostic of mitral constriction, excepting a few cases of aortic regurgitation, to which reference will be made in the discussion of that lesion. Auricular pulsation may occasionally be felt.

The pulse of mitral stenosis is necessarily small, since the normal quantity of blood does not enter the left ventricle. The rhythm may be perfectly regular as long as compensation is maintained, but becomes decidedly irregular as soon as cardiac power fails. The sphygmogram shows the irregularity as well as the general weakness of the pulse, and certain imperfect systolic efforts too weak to be appreciated by the touch.

FIG. 14.



Sphygmogram in Mitral Obstruction, from a man aged 30 years. Dyspnœa, cough, slight blueness of lips and fingers.

Percussion.—Cardiac dulness increases more in an upward direction and to the right of the sternum than toward the left. When the left auricle is much dilated dulness rises quite high along the left border of the sternum, and the right ventricle may be found in extreme cases extending almost or quite to the right nipple-line. In cases in which pulmonary collapse or compression has occurred I have seen complete dulness of the left lung, and have even known the condition to be mistaken for the consolidation of phthisis.

Auscultation.—In typical cases there may be heard, just before the first sound, and ending in this, a prolonged and rumbling murmur, commonly known as the presystolic murmur. This murmur is produced during the passage of the blood from the auricle to the ventricle. It begins in the latter part of the auricular systole, and ends in the sharp first sound, which indicates the beginning of ventricular contraction. The time of the murmur is therefore diastolic, but as it nearly always occurs in the latter part of this period and continues up to the ventricular systole, the name "presystolic" is not inappropriate. Much discussion has taken place regarding the manner of production of this murmur and the exact stage of the cardiac revolution during which it occurs, but the view above expressed is both the simplest and the most reasonable. The presystolic murmur is absent in some cases from beginning to the end, and very commonly disappears when compensation fails. It may occur without the palpable thrill, or the latter may be detected when the murmur is absent. Sometimes it is inaudible at the apex, but heard distinctly above and to the left of this point. Flint first called attention to a peculiar presystolic murmur found at the apex in certain cases of aortic regurgitation, but the existence of a diastolic murmur at the aortic cartilage and of the rational symptoms of aortic insufficiency serve to arouse suspicion in these cases, though a positive decision as to the nature of the murmur may sometimes be impossible.

The sharpness of the first sound following immediately after the rumbling murmur is also characteristic. The cause of this phenomenon is difficult to determine, but it seems probable that sudden closure of the tricuspid valve, resulting from hypertrophy of the right ventricle, plays an important part. The sudden first sound may be found in cases in which the murmur has not been present or has disappeared, and under such circumstances is highly significant. I have noted also, occasionally, as showing that regurgitation may coexist with obstruction, a typical presystolic murmur running up to a distinct systolic murmur, which partially concealed the first sound. Occasionally there is splitting or reduplication of the first sound at the apex.

The second sound at the pulmonic cartilage is loud, and often ringing in character. Reduplication of the second sound is much more frequently noted than that of the first, and it has, therefore, a definite amount of diagnostic value. Its cause is readily found in the excessive tension in the lesser circulation, the hypertrophy of the right ventricle, and the want of exact correspondence in the systole of the two ventricles, and consequently of their respective arterial outlets. When cardiac compensation has failed, the auscultatory signs become confused by the excessive rapidity and irregularity of the heart's action, by the sharpness of the first sound, and the disappearance of the murmur.

A double sound, like that described by Traube in aortic insufficiency, is occasionally heard in the femoral artery.

Aortic Insufficiency.

Aortic regurgitation stands next to mitral insufficiency in frequency. It is more common in men than in women. Among the causes which specially lead to it are alcoholism, syphilis, and muscular strain, the valvular disease being closely allied to atheroma of the aorta, and not rarely secondary to this condition. More rarely, insufficiency of the aortic valves results from rheumatic endocarditis, and in these cases the patient may be quite young. The atheromatous form commonly occurs in persons over forty or fifty years.

The valvular lesion consists in various forms of contraction, puckering, or curling of the segments, with increasing sclerosis, causing rigidity. Later the occurrence of calcification still further stiffens and disables the valve. In rare cases insufficiency is brought about by adhesion of the segments to the intima of the aorta, by ulceration, or by traumatic rupture of one of the segments. In long-standing cases the valves may be found reduced to mere stumps or calcified projections at the orifice. Not rarely a certain amount of stenosis coexists, when the leaflets are united or project inward as rigid elevations; but simple regurgitation is much more frequent than simple stenosis.

Relative aortic insufficiency may result from great dilatation of the aorta, as in aneurism, when the normal valves are no longer able to close the orifice. The dilatation may in some cases be so great that the segments are drawn apart at their edges of insertion, and stretched or made tense along the unattached margin. Usually, however, the valves are also diseased.

Congenital insufficiency is very rare. When two of the segments are united into one, the valve may still be competent, and usually is so; but this condition predisposes strongly to subsequent valvular disease.

The regurgitation of the blood from the aorta into the left ventricle, with the normal supply coming from the auricle, causes considerable overfilling of the ventricle at each diastole, and therefore dilatation of its cavity results. Later, hypertrophy of the wall occurs, and with the existing dilatation gives rise to enormous enlargement of the heart, the *cor bovinum* of the older writers. In no other condition do hypertrophy and dilatation reach the same degree. The muscular substance is firm and greatly thickened, the ventricular cavity much enlarged, and the trabeculæ and papillary muscles flattened by the increase of intracardial pressure. Usually there is associated disease of the aorta and general arterial system. The ascending portion of the aortic arch is dilated, and the intima presents more or less advanced sclerosis and atheroma. The coronary orifices may be occluded by atheromatous deposits, or these vessels may be atheromatous throughout, and in either case cardiac degeneration results. Interstitial myocarditis may be found in localized areas or may be diffuse, and in the later stages cloudy swelling and fatty degeneration indicate the further interference with the heart's nutrition. Excessive dilatation of the ventricle may supervene, and lead to relative insufficiency of the mitral segments. The increased pressure in the ventricle causes dilatation of the left auricle, and some hypertrophy of the right ventricle, even when the mitral valve is competent, but these conditions reach their maximum after the establishment of relative insufficiency of the mitral leaflets. The increased strain upon the mitral valve frequently induces slight sclerotic changes, which are recognized in the nodular thickening of the edges; but mitral regurgitation is more frequently relative in nature.

The general arterial sclerosis associated with aortic regurgitation often reaches a high grade, and is doubtless to a certain extent attributable to the great and sudden distention of the arteries at each contraction of the powerful ventricle. This arterial disease, together with the ventricular enlargement, in turn accounts for the tendency to cerebral hæmorrhage and bleeding in other places. The compensation of aortic insufficiency is often maintained for years, and is more nearly perfect than in any other valvular lesion; but when myocardial degeneration supervenes, the cardiac power is apt to diminish with great rapidity.

Physical Signs.—*Inspection.*—The impulse of the heart is displaced to the left and downward, and may be seen in the sixth or seventh interspace between the mammary and anterior axillary lines. It is usually strong and heaving, though diffuse pulsation frequently accompanies it. The latter is to be explained by the associated dilatation. The whole præcordial region may be distended, and may pulsate with each ventricular systole. The vessels of the neck are usually seen to pulsate violently, and in more decided cases the temporal, the brachial, and all of the superficial vessels throb forcibly. I have more than once known an unsuspected case to be discovered by the throbbing of the retinal vessels, seen on ophthalmoscopic examination. The dilatation of the vessels is quick and decided, and may cause swelling of the tissues of the neck at each ventricular systole. Sometimes on rubbing the forehead or pressing gently on one of the finger-nails alternate blushing and paling may be observed. This phenomenon, to which Quincke gave the name of "capillary pulse," is highly significant, though it is not confined to aortic regurgitation. Even pulsation of the peripheral veins of the foot and hand has been observed. This appears as a progressive venous pulse flowing centrally.

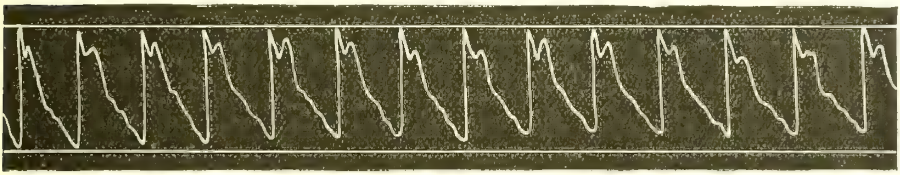
Palpation—The force of the impulse is decided and heaving in character. In addition there may be general pulsation of the whole præcordia, and sometimes a diastolic thrill is felt at the base of the heart and at the aortic cartilage. The occasional occurrence of a presystolic thrill, similar to that of mitral stenosis, at the apex has been referred to in the description of the latter. It may be due to the fact that during diastole the mitral leaflets are floated out by the regurgitating blood, and at the same time receive a fluttering motion from the direct current coming from the auricle. Distinct pulsation is sometimes noted in the second right intercostal space, resulting from dilatation of the aorta, and has been mistaken for aneurism of the aortic arch.

The pulse of aortic insufficiency was first accurately described by Corrigan, by whose name it is frequently designated. It is peculiarly quick or jerking as it strikes the finger, and often conveys an impression of good size and force, but immediately falls away or collapses. These characters are often made more striking by elevating the patient's arm above his head, when the pulse is felt. They are readily explained by the excited action of the ventricle and the low tension existing in the arteries on account of the regurgitation. The pulse is therefore easily compressible, though forceful at its impulse. The names by which, in addition to that of Corrigan, it is known—water-hammer pulse, collapsing pulse, and pulse of unfilled vessels—are sufficiently descriptive of its character. It is usually somewhat accelerated and regular until compensation fails. The sphygmographic tracing illustrates by its sudden rise and fall the palpable qualities of the pulse. (See Fig. 15.)

Palpation of the carotid vessels discovers similar pulsation, and sometimes a rough arterial thrill. Pulsation and thrill in the suprasternal notch has sometimes led to the mistaken diagnosis of aneurism. Indeed, when aortic valvular disease is associated, as is not rare, with atheroma of the aorta, leading to thickening and enlargement, the question of aneurism must be considered critically.

Reference has been made to the occasional occurrence of venous pulses, but they are rarely observed, and almost never become sufficiently distinct to be palpable. A systolic swelling or pulsation of the liver, arterial pulsation of the liver, is occasionally noted, and resembles closely the venous pulsation of tricuspid regurgitation. Pulsation of the spleen is very rare.

FIG. 15.



Sphygmogram of Aortic Regurgitation, from a man aged 60 years. Anæmia, dyspnœa, vertigo, cardiac pain, slight œdema.

Percussion.—The enormous hypertrophy of the left ventricle causes lateral and downward extension of the cardiac dulness, and in the later stages there may be increase of dulness upward along the left border of the sternum and to the right when the left auricle and right ventricle have become enlarged. The roundness of the apex, as determined by percussion, will indicate the amount of dilatation associated with the hypertrophy. In addition to the palpable and visible pulsation at the aortic cartilage the dilated aorta, especially if its walls are thickened by atheroma, may cause a certain amount of dulness under the manubrium and extending somewhat beyond its left border.

Auscultation.—The characteristic murmur of aortic incompetency is a prolonged, soft or loud, and diastolic bruit, heard with greatest distinctness in the second intercostal space of the right side or under the manubrium, and transmitted strongly downward toward the ensiform cartilage, while at the same time it is propagated somewhat along the aorta and great vessels. The character of the sound varies widely in different cases, being often soft, sometimes quite harsh, and occasionally musical. In a case of traumatic rupture of one of the aortic segments which came under my observation many years since there was a loud, whirring murmur, audible at a considerable distance from the patient. In ordinary non-traumatic cases the murmur may be occasionally heard all over the thorax and in the vessels of the neck. This murmur is highly significant of aortic regurgitation, though a similar bruit may occur in aortic aneurism, and in the rare cases where there is a communication of the aorta with the pulmonary artery through an aneurism or patulous ductus Botalli. It may completely or only partially replace the second sound at the apex, but in a former case a second sound is sometimes audible in the vessels of the neck. Sometimes the murmur is heard with greatest intensity at the apex—a fact which Balthazar Foster considered indicative of failure of the left aortic segment, by which the regurgitant blood-stream would be directed upon the anterior mitral leaflet, and then to the apex of the ventricle.

The first heart-sound is often noticeably weak. Secondary murmurs may be developed at the apex and at the aortic cartilage. In the former situation a systolic murmur of mitral regurgitation denotes the increasing ventricular dilatation and relative insufficiency of the valve. A presystolic murmur, to which reference has already been made in the description of Mitral Stenosis, is occasionally present, and is explained in the same manner as is the presystolic thrill above described. It is, however, a rare occurrence, though Lees has collected fifteen cases, including his own, in which autopsy revealed aortic regurgitation and not mitral stenosis. A systolic murmur in the second right interspace, and transmitted into the carotid arteries, may be indicative of associated aortic stenosis or of mere roughening of the valves or intima of the aorta; but the latter conditions are much more frequently present than is actual stenosis.

Auscultation of the femoral artery sometimes detects a double sound (Traube), but more frequently a double or to-and-fro murmur, in place of the single systolic murmur discovered in healthy persons when firm pressure is made with the stethoscope. This double murmur of Duroziez is heard in a considerable proportion of the cases, and has a certain confirmatory value in diagnosis. Sometimes there are three murmurs—two with the systole and one diastolic.

Aortic Stenosis.

Pure aortic stenosis is a rare condition. Nearly always there is also regurgitation, and in the development of the latter moderate stenosis is often associated. It is most frequently produced by slow, atheromatous processes affecting the valves and root of the aorta in men advanced in years. The valve-segments become more and more rigid and calcareous, projecting inward and obstructing the lumen of the orifice. Sometimes, as a result of rheumatic endocarditis, the leaflets are united and obstruction exists in younger persons. An extreme instance of this kind was recently in my wards at the University Hospital in the person of a boy of fourteen years. Very rarely the lesion is congenital. Stenosis of the orifice without disease of the valves may occur when the valvular ring is infiltrated and calcareous. Dilatation of the aorta may accompany aortic stenosis, but is much less frequent than in regurgitation.

The first effect of narrowing of the aortic orifice and obstruction to the outflow of blood is hypertrophy of the left ventricle, which maintains the proper fulness of the arterial system, and for a long time may be unaccompanied by dilatation. In the later stages, however, dilatation of the heart accompanies the hypertrophy, but neither condition reaches the enormous proportions seen in insufficiency. The mitral valves may be slightly thickened from the constant strain in closing, and, when dilatation of the ventricle is marked, relative insufficiency may develop. In the latter case, but to a lesser degree even without it, the pressure within the left auricle is increased and dilatation and hypertrophy supervene. Hypertrophy of the right ventricle is a late manifestation of increasing obstruction to the pulmonary circulation.

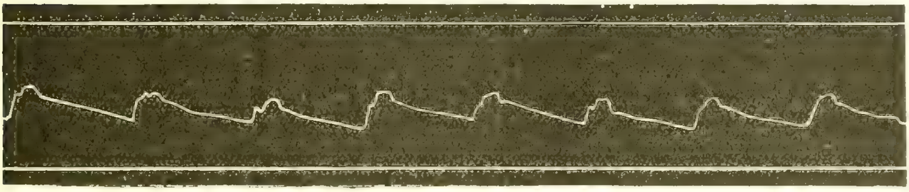
In most cases there are decided arterial degenerations associated with the cardiac lesion or antedating the latter. These are most marked in the arch of the aorta, near the valves, and often lead to narrowing of the orifices of the coronary arteries. The latter vessels are, however, themselves involved in many cases, and cardiac degenerations are therefore prone to be developed.

Physical Signs.—*Inspection.*—The apex of the heart is displaced to the left and downward, though not to the extent seen in aortic regurgitation. The the impulse is localized and often apparently weak.

Palpation.—The apex-beat is weak and slow. This is explained by the protracted systole, which deprives the impulse of its usual sharpness and suddenness, but may be regarded as a compensatory condition, in permitting a greater time for the outflow of blood through the narrowed orifice. A systolic thrill is frequently detected at the base and in the second right intercostal space. It may be very decided, and occasionally is also felt in the suprasternal notch and in the cervical vessels.

The pulse typical of aortic stenosis is small and infrequent, with a peculiar hesitating or halting character. These qualities are readily explained by the narrowing of the orifice and the slow contraction of the ventricle. As long as compensation is maintained the pulse is usually regular, and it may be so to the end. The sphygmogram shows a gradual ascent and descent of the curve, which is rarely high. An anacrotic notch with marked tidal wave and gradual descent are characteristic conditions, which may prove of value, as in the case from which the accompanying tracing was obtained, in deciding that the murmur heard is in reality indicative of stenosis at the orifice. General arterio-sclerosis, however, produces a very similar pulse and sphygmogram.

Fig. 16.



Sphygmogram of Aortic Stenosis, from a man aged 60 years. Pulse-rate 50. Anæmia, vertigo, tinnitus aurium, headache.

Percussion indicates the degree of hypertrophy of the left ventricle. Dilatation of the aorta may be manifested by dulness under the manubrium sterni and slightly to the left of it.

Auscultation.—A long-drawn, systolic murmur is heard in the second intercostal space at the right border of the sternum, and is usually transmitted to the vessels of the neck. The first heart-sound is obscured by the murmur at the aortic region, but is plainly heard at the apex. The second aortic sound is weak, and may be absent, from the fact that the stiffened valves are incapable of sharp closure. Very commonly a diastolic murmur of regurgitation also becomes audible, and a double to-and-fro murmur is very characteristic. Few cases of stenosis occur in which there is no regurgitation, but the clinical features of the case will depend upon the relative preponderance of the one or the other. A systolic murmur of similar locality and distribution may occur in roughing of the aortic valves or of the intima of the aorta, in aneurism of the ascending part of the arch, and in conditions of great anæmia. The discrimination of these will depend upon the study of the pulse, on the presence or absence of hypertrophy, and upon the individual symptoms of the case; but it is not possible in all instances to make a positive diagnosis. Tumors and enlarged lymphatic glands may constrict the root of the aorta and produce the same pathological results and clinical manifestations as are seen in aortic stenosis. Lemoine has recently described cases in which a presystolic murmur of rumbling character, similar to that of mitral stenosis, was present at the aortic region, in addition to the usual systolic murmur, and explains the condition by assuming the existence of a dilated conus arteriosus in which the blood is agitated prior to its expulsion through the narrowed orifice.

TRICUSPID INSUFFICIENCY.

Tricuspid regurgitation may be organic or relative, the latter being much more frequent than the former. Actual disease of the tricuspid valves with regurgitation is a not uncommon result of fœtal endocarditis. In a notable case reported by Peter a rough systolic murmur replaced the first sound of the fœtal heart, and subsequent examination of the stillborn child disclosed vegetations and thickening of the tricuspid leaflets, with retraction of the segments by the chordæ tendineæ. After birth chronic endocarditis and insufficiency of the tricuspid valve are most frequent in children, diminishing in frequency as age advances, though I have frequently noted slight nodular thickening of the edges of the segments in persons dead of phthisis and other pulmonary disease. Byron Bramwell found the tricuspid valve affected in 50 per cent. of a series of cases of simple acute endocarditis, and suggests that the rarity of sclerotic changes is due to the subsidence and complete cure of the lesions on this valve, probably because of the lesser strain to which it is subjected. Very rarely one of the tricuspid segments may be ruptured by strain.

Relative insufficiency of this valve is, on the contrary, a common condition in stenosis or regurgitation at the mitral valve and in such obstructive lung diseases as phthisis and emphysema. In these cases the right ventricle first undergoes hypertrophy, which maintains the circulation through the lungs, but later, with increasing tension of the lesser circulation, the ventricle dilates and leakage at the tricuspid orifice results. Finally, dilatation of the right auricle, the venæ cavæ, and the peripheral veins gives rise to general cyanosis and leads to a fatal termination. The right ventricle is incapable of further hypertrophy to compensate the regurgitation, such as we have seen in cases of mitral insufficiency, because the tricuspid failure is the result of advancing dilatation, occurring after hypertrophy of the right ventricle has been taxed to its utmost compensatory power to maintain the pulmonary circulation. Dilatation therefore becomes more and more pronounced, and in an extreme case of mitral stenosis I have seen the right ventricle so enlarged and its walls so thinned that it presented the appearance of a globular tumor, through whose membranous covering the dark blood could be perceived. The right and left auricles were enormously enlarged, and the venæ cavæ two inches in diameter.

Physical Signs.—The physical signs will vary largely with the nature of the primary condition upon which the tricuspid insufficiency is dependent.

Inspection.—The apex of the heart may be displaced to the left if mitral regurgitation has preceded the tricuspid lesion. In cases of mitral stenosis the impulse may be in its normal position or moderately displaced by the increasing dilatation of the right ventricle. Epigastric pulsation and bulging of the lower sternal and epigastric region, or even distinct epigastric tumor, may be noted, and I have seen these signs develop very suddenly in a case of old mitral regurgitation during an intercurrent attack of influenza. They are dependent upon the dilatation of the right ventricle and the congestive enlargement of the left lobe of the liver. The apex of the left ventricle may be displaced from the chest-wall by the dilated right ventricle.

Pulsation of the jugular veins is a characteristic sign. It may at first be confined to the lower portion of the vein by the valve existing at that point, but later the whole vein is seen to pulsate strongly with each ventricular systole. Sometimes the distended cervical veins receive a communicated pulsation from the underlying carotid arteries, but this is readily distinguished by pressure on the lower part of the vein, which does not affect the spurious pulsation, while it prevents the true venous pulse above the point of pressure. In some cases a double pulse is seen, the explanation being that the weaker systole of the right auricle and the stronger contraction of the ventricle each produce pulsation proportioned to their strength. The phlebogram may show this condition in an anacrotic notch.

Palpation.—Epigastric pulsation is usually distinct, and there is often systolic pulsation of the liver. The latter is enlarged, and its edge may be felt below the margin of the ribs, sometimes as low as the umbilicus. With each contraction of the right ventricle there is an expansile swelling of the liver, which may be so distinct as to be readily visible. Similar pulsation of the spleen is less commonly observed. A systolic thrill may occasionally be discovered over the right ventricle. The impulse of the apex of the heart may disappear when the dilatation of the right ventricle displaces the left heart from the wall of the chest.

The condition of the pulse depends upon the antecedent lesion of the valves of the left ventricle. It is usually rapid and irregular. Popoff has recently called attention to the greater weakness of the pulse of the right wrist, which he explains by the pressure exerted on the innominate artery by the enlarged right auricle and vena cava.

Percussion.—The extension of dulness to the right of the sternum and downward toward the epigastrium is the most marked feature on percussion.

Auscultation.—A systolic murmur heard at the lower part of the sternum and near the ensiform cartilage is usually present, though marked regurgitation with pulsating veins is sometimes unaccompanied by any murmur. The murmur is soft or harsh, and often musical or whistling. In many cases it is peculiarly superficial. The first sound is obscured where the murmur is loudest, but at the apex may be distinct; and the second sound, which had previously been accentuated as a result of mitral disease, loses in sharpness as soon as the tricuspid insufficiency is established. The presystolic or systolic murmurs of antecedent mitral stenosis or regurgitation may be heard at the apex or slightly to the left of it; but, as in the cases alluded to before, the events occurring in the left heart may be inaudible until the interposed right ventricle has become less distended and the left ventricle has resumed its position immediately under the chest-wall. It may be difficult to decide whether a systolic murmur heard at the ensiform cartilage is transmitted from an existing mitral murmur or due to tricuspid regurgitation; but the character of the sounds is sometimes quite different, and a mitral murmur is rarely transmitted so far to the right as is the tricuspid. When the right ventricle is enlarged and pulsation of the jugular veins and liver coexists, little doubt can be entertained as to the presence of insufficiency of the tricuspid valve.

Auscultation of the lower part of the jugular vein sometimes reveals a venous sound due to the closure of the valve at this situation, but later the valve becomes insufficient, and the sound is no longer heard. A similar valvular sound has occasionally been noted in the femoral vein.

TRICUSPID STENOSIS.

Tricuspid stenosis is a very rare form of valvular disease. It was formerly looked upon as invariably congenital, but probably in at least an equal number of instances arises after birth. In a minority of the cases an antecedent history of rheumatism is obtained. It is more common in women than in men, and generally is not detected before the twentieth year. Leudet analyzed 114 collected cases, and Bedford Fenwick 46. In the statistics of the former 80 per cent. occurred in women, and in one-third the history of rheumatism was undoubted. Of Fenwick's cases, 38 were women and but 8 were men, while rheumatism had occurred in one-half of all.

Stenosis of the tricuspid orifice is generally found associated with other valvular disease, though a few cases have been observed in which no such association existed. The most frequent combination is with mitral stenosis, though aortic regurgitation not rarely coexists.

The valves are found to be thickened and stiff and the neighboring segments united. In a rare case, reported by Gairdner, a globular tumor of the auricle, acting like a ball valve, caused obstruction at each auricular systole. The stenosis at the tricuspid orifice leads to dilatation of the right auricle, and later to general venous repletion. The right ventricle is hypertrophied when the coexisting mitral stenosis causes increase of tension in the pulmonary circulation.

The physical signs are rarely definite or decided. Enlargement of the right auricle may be detected by percussion, and there is sometimes a palpable presystolic thrill over the right ventricle. The latter may occasionally be separated from a coexisting mitral presystolic thrill by an area in which palpation furnishes no signs; but more commonly the two are merged and inseparable. A presystolic murmur, ending in a sharp first sound, heard over the lower part of the sternum, is characteristic when unassociated with other murmurs; but it is usually impossible to distinguish this murmur from that of mitral obstruction; and confusion may arise from the coexistence of an aortic diastolic murmur. Altogether, the diagnosis cannot be made with certainty excepting in the rarest instances.

PULMONARY STENOSIS.

Pulmonary stenosis is very rarely found, except as a result of congenital malformation or fœtal endocarditis. In the few recorded cases in which the lesion seemed to have been developed after birth, contraction of the valves, with union of the segments, has been noted, together with vegetations of acute endocarditis. Sometimes the conus arteriosus may be narrowed by contraction following myocarditis. The congenital form will be described with the malformations of the heart, of which it is a frequent and important example. Hypertrophy of the right ventricle compensates by forcing the blood through the narrowed orifice, thus maintaining the circulation through the lungs.

The **physical signs** are unreliable. A systolic murmur is usually heard in the second left intercostal space near the sternum. It is harsh and superficial, and rarely transmitted to any great distance. The frequency, however, of anæmic *bruits* and pulmonic murmurs, due to pressure from without, makes the diagnosis uncertain. The murmur of aortic stenosis is readily excluded by its transmission into the cervical vessels. The second sound is weak, from the diminution of pulmonic tension. A systolic thrill is noted in a considerable proportion of the cases.

PULMONARY INSUFFICIENCY.

Pulmonary regurgitation is extremely rare. It may be congenital, or it may result from endocarditis after birth. The valves have sometimes been found sclerosed and rigid or adherent to the wall of the artery. In congenital cases two of the segments may be united, and the original line of separation is sometimes no longer demonstrable. Relative insufficiency has been noted in cases of aneurism of the pulmonary artery, and, according to Steell, in mitral stenosis.

The right ventricle undergoes hypertrophy and dilatation, and when the latter becomes excessive relative insufficiency of the tricuspid valves is developed.

The **physical signs** are uncertain. A diastolic murmur may be heard with greatest distinctness in the second right intercostal space, and is transmitted to the lower part of the sternum. It is difficult to distinguish this murmur from that of aortic regurgitation, though the enlargement of the right ventricle, the establishment of tricuspid insufficiency and early cyanosis, and the absence of the water-hammer pulse would furnish strong indications of pulmonic disease. In addition, it may be noted that the pulmonary murmur is loudest to the left of the sternum, the aortic to the right.

COMBINED VALVULAR LESIONS.

In nearly a half of all cases of valvular disease more than one lesion is found. There may be either a double lesion, obstruction and insufficiency, at a single valve, or single or double lesions at different valves. Mitral regurgitation with aortic regurgitation and obstruction is the most frequent combination. Mitral stenosis with regurgitation comes next in point of frequency. It is probable that in nearly every case of obstruction of the mitral valve in which the segments themselves are diseased, some regurgitation also exists. The frequency of relative tricuspid regurgitation at the termination of mitral disease has been referred to.

In many cases the development of a secondary relative insufficiency is compensatory in nature. Thus in aortic regurgitation the occurrence of mitral leakage prevents overfilling of the left ventricle, and probably prevents a fatal distention.

The **physical signs** are those of the separate lesions combined, and the **diagnosis** will depend on the careful study of the murmurs present. In many cases it will be found that, though there are several valvular imperfections, one only is of a serious nature; and it will be necessary to determine this fact by careful examination of the secondary changes in the heart and other organs.

Symptomatology of Valvular Disease.—The clinical course of valvular disease of the heart is conveniently and naturally divided into the stage of established compensation and that of failing or broken compensation, or, as French authors designate them, the stage of eusystole and that of hyposystole or asystole.

During the former of these stages the symptoms may be most obscure, and not rarely the patient considers himself in complete health. Physicians are frequently startled by the discovery of a decided valvular lesion in individuals suffering with some trifling acute ailment, and examiners for life insurance often reject on account of a valvular lesion persons who had no suspicion of its existence. Such complete compensation and latency of symptoms is not confined to the lesions of any particular valve, but seems, on the whole, more frequent when the aortic valve is affected. In most cases, however, careful inquiry will disclose the existence of some symptoms, perhaps trifling in nature, dating from the time of an attack of acute rheumatism or some infectious disease, or they may have been present for years without having any definite time of origin. The patient will state that he has noticed a slight oppression of breathing when walking more rapidly than usual, when mounting stairs, or lifting weights. Even more trifling conditions may have affected him, such as the oppression of a crowded room or the least emotional excitement, and at the same time he may have noted increased rapidity of the pulse or have been conscious of the beating of his heart. The pulse is usually regular, but may be constantly irregular, though compensation is perfectly maintained. Sometimes there is the feeling of weight or discomfort at the heart, and in rare cases occasional attacks of cardiac pain. The patient is easily fatigued, may take cold easily, and suffers with gastric disturbances after slight indiscretions in diet. His general health is not impaired, but he feels that his customary vigor and power of endurance have suffered some change. The complexion may be poor, and sometimes a little blueness of the lips and finger-nails is observed; or pallor and anæmia may occur instead. These symptoms for the most part are due to the fact that the compensatory power of the heart is sufficient to maintain the proper circulation under ordinary conditions, but incapable of combating with special emergencies.

When, as a result of intercurrent diseases, chiefly the fevers, or of advancing valvular and arterial disease, the nutrition of the heart fails, cardiac degeneration and failure of the compensation result. The pulse now becomes more rapid, and is often irregular in rhythm; there is decided palpitation, and the patient suffers dyspnœa even to the most extreme degrees. In addition, the local subjective sensations of oppression or constriction and pain become more pronounced, and the evidences of failing circulation in various parts of the body, and especially in the internal organs, become decided. The complexion becomes dusky, the lips, the ears, the fingers, or the whole surface may grow cyanotic, and dropsy of the subcutaneous tissues, and later of the serous cavities, gives evidence of the failing circulation. In some cases the patient comes to the physician complaining of vertigo, syncopal attacks, or constant headache with throbbing of the vessels; in others dyspepsia, disordered condition of the bowels, and uneasiness in the region of the liver may indicate the disturbance of the abdominal circulation.

The onset of the symptoms of failing compensation may be abrupt or gradual. The latter is more common, but the former is frequently observed when persons having a chronic valvular defect are ill with infectious fevers, such as typhoid fever, diphtheria, or influenza. An example of the extreme acuteness of onset of cardiac dilatation and broken compensation was recently admitted to my wards at the University Hospital. The patient, a man of sixty years, had an old regurgitant murmur at the mitral valve, and became ill with influenza, during which bronchitis was pronounced. Very suddenly in the course of the disease the heart became extremely rapid and irregular, the surface became cyanosed, and universal œdema was developed. The liver was much enlarged; there was undulation of the jugular veins; there were pulsation and tumor in the epigastrium; and a loud regurgitant murmur was detected in the tricuspid region. In this case undoubtedly the right ventricle had suddenly dilated, permitting tricuspid regurgitation, and leading to the general distention of the veins and development of dropsy. On the other hand, it will be found in a large number of cases that the ease with which compensation is disturbed gradually increases until the circulation can no longer be maintained, and pronounced symptoms of failure of the cardiac power supervene.

It is impossible to draw sharp lines separating the individual valvular lesions from one another, nor can a clinical course be sketched which will apply to all cases of a particular form. It will therefore be best to describe the symptoms without reference to the nature of the lesion, further than to indicate the lesion which is most apt to be present when the symptom under discussion is noted.

Subjective symptoms form an important part of the clinical manifestations of all cases of valvular disease. The first of these to be noted is shortness of breath, or dyspnœa, which results from obstruction to the pulmonary circulation, and is therefore developed earliest and reaches the highest grades in insufficiency or stenosis of the mitral valve. As long as compensation is fully maintained there may be no dyspnœa until some unusual exertion or excitement has taxed the cardiac power; but in many cases of mitral diseases there is constant shortness of breath, without any other sign of failing compensation. When compensation is lost, dyspnœa becomes a distressing symptom: the patient pants for breath; the slightest weight upon the chest oppresses him; he is no longer able to lie recumbent, and sleep is seriously disturbed. The extreme dyspnœa and orthopnœa of the later stages results not only from the failing circulation, but also from the secondary changes occurring in the lungs. To these may be added the increased vascular tension and the blood-contaminations resulting from renal and peripheral engorgement, with defective elimination and metabolism. The congestion of the pulmonary vessels soon leads to the development of serous transudation and œdema of the lungs, and in protracted cases to cyanotic induration. Still greater interference with the pulmonary circulation and the breathing occurs when pulmonary thrombosis and hemorrhagic infarctions have supervened, or when the lung is compressed by the dilated heart or by hydrothorax. In the instructive case of Friedreich, before referred to, the dilated auricle in mitral stenosis almost completely obliterated the lumen of the left bronchus by its pressure. Sometimes dyspnœa has a peculiarly paroxysmal nature, to which the term "cardiac asthma" is strictly applicable. In these cases the patient is suddenly seized with great shortness of breath, which lasts for variable periods of time and gradually subsides. Such attacks may develop even in cases in which the compensation is fully established, and may recur at irregular intervals for years. There is in such subjects an associated weakness and irritability of the respiratory muscles and nerves; and over-exertion, excitement, exhausting reflex irritation from a disordered stomach, or acute bronchitis from exposure may precipitate a crisis.

In the very latest stages Cheyne-Stokes breathing or rhythmical dyspnœa is occasionally noted as a terminal symptom. It is more frequently present in aortic disease and when the myocardium has suffered considerable degeneration, and renal complications, so often noted in valvular disease, increase greatly the tendency to this phenomenon. The peculiarity of this condition consists in the alternation of periods of breathing with periods of cessation. After a pause in the respiration the patient begins to breathe, almost imperceptibly at first, then more and more deeply and noisily until a climax is reached, when the efforts again decline, and finally end in complete cessation. The pause or period of apnœa varies in length from a few seconds to a half minute or a minute, and terminates with the beginning of shallow respirations. During the apnœic period the patient's pupil grows smaller and the pulse less rapid. In rare cases consciousness, absent as a rule, is temporarily regained when the respiratory efforts reach their climax. The explanation of this peculiar form of breathing remains in doubt, though some disturbance of the respiratory centre in the medulla doubtless plays a part in its production. It is not unusual in meningitis and cerebral hæmorrhage and in uræmic coma.

Subjective symptoms referred to the heart are rarely absent. At first there is merely the feeling of unnatural fulness or distention in the region of the heart, or a vague sense of oppression, likened to a constriction or a weight upon the chest. When, however, the patient exerts himself or becomes excited, the feeling of palpitation is developed and grows pronounced as compensation fails. The impulse of the heart is usually forcible, but the sense of palpitation may be present in cases in which the heart's action is extremely weak, and absent when it is most pronounced. The pulse-rate keeps pace with the increased action of the heart. It is usually more or less irregular, though perfect rhythm is sometimes maintained to the last.

Irregularity of the pulse is more common in mitral than in aortic disease, and, especially in mitral stenosis, frequently reaches an extreme degree. As a rule, cardiac arhythmia may be taken as an indication of failing compensation, but there are cases in which such is not the case. A seeming irregularity may result from occasional weakness of the systole, when the corresponding pulse-wave fails to be perceptible at the wrist, but generally where such inequality of contraction prevails the rhythm is likewise disturbed. The pulse should be counted at the wrist while the ear is noting the cardiac action, and the sphygmograph is of signal value in recording contractions which are too feeble to be felt. Instead of frequency, there is sometimes unusual slowness and infrequency of the pulse, especially in aortic stenosis. This has already been mentioned as of a certain compensatory value in giving time for expulsion of a proper quantity of blood through the narrowed orifice. It is not unusual in these cases to find a rate of 60 or 50 beats per minute, and when interstitial myocarditis is present the infrequency may become even more decided. On the other hand, excessive rapidity of the pulse-rate—a symptom to which the name tachycardia has been applied—may occasionally be present, though it more frequently results from other causes. The palpitation of valvular disease, as a rule, subsides to a large extent soon after the patient is placed at rest physically and mentally, whereas in tachycardia a rate of 160 or 200 may be maintained uninterruptedly for long periods of time. It is difficult to separate high grades of palpitation from what we designate as tachycardia, since the sense of palpitation and dyspnœa are sometimes, though not, as a rule, present in the latter. The excessive rapidity of tachycardia is, however, rarely attained in palpitation, and the subjective sensations of the latter are often absent in the most pronounced instances of tachycardia. The symptom is more common in mitral than in aortic lesions.

Pain at the heart is much more frequently noted in aortic regurgitation or stenosis than in mitral disease. Sometimes it may be merely an intensification of the feeling of constriction at the præcordium; in other cases the most violent attacks of darting pains in the region of the heart and radiating to the arms may be observed. Not rarely these anginoid pains assume the character of true angina pectoris. The pain more frequently radiates to the left than to the right arm, and may be attended by blanching of the surface and a more or less persistent numbness of the skin. It is especially in cases of atheromatous valvulitis and aortitis, with high tension within the artery or ventricle, that such paroxysms are experienced, and it has seemed to me that the mechanical effect of hypertension has much to do with the occurrence of the pain. The associated myocardial degeneration seems also to play a part, since similar manifestations are encountered in acute or chronic degenerations of the heart without involvement of the valves. Cases are frequently encountered where anginoid pain is developed regularly by a definite amount of exercise. In some subjects it is only brought on by brisk walking after meals; in others the tendency is so great that gentle exercise develops it. The pain grows more intense until the patient rests, when it rapidly subsides. I have often spoken of this as mechanical angina. It is associated with atheroma and thickening of the base of the aorta, with or without valvular disease. It is more fully described in the section on Angina Pectoris.

In cases of mitral disease, more commonly than in aortic, there is frequently abdominal pain, associated with congestion of the liver or portal circulation. Generally this consists of vague oppression or pain in the epigastrium or under the lower ribs of the right side, and is often associated with abnormal pulsation or tumor. In rare cases it becomes quite severe, and may prove an urgent symptom. Severe colicky pain with distention of the abdomen and diarrhœa, sometimes hæmorrhagic in nature, results from embolism of the mesenteric artery. It is an unusual symptom, and more apt to occur when there are fresh vegetations of acute endocarditis upon the valves. Many years ago, before Koch's discovery, I attended a case of chronic mitral valvulitis with that eminent clinician, the late J. Forsyth Meigs. The hectic symptoms were so intense that the scattered centres of destructive disease of the lungs were considered tuberculous, and the frequent recurring attacks of severe abdominal pain with diarrhœa seemed to indicate tuberculous ulceration of the bowel. The rarity of phthisis combined with valvular disease of the heart is well known. The autopsy showed suppurative changes and several large hæmorrhagic infarcts of the lungs, and as many as twenty separate embolisms of branches of the mesenteric arteries, each of which had caused aneurismal dilatation, filled with clots as large as a cherry. An ill-defined aching in the back may be present when the kidneys are congested; and painful affections of the joints, even with distinct swelling and with fever, have sometimes been noted.

In uncompensated valvular disease the symptoms discovered by examination are largely due to failing circulation in the internal organs and in the peripheral vessels.

The appearance of the patient's skin may give the first indication of the venous stasis in the development of cyanosis. At first there is merely a slight duskiness of the lips, the ears, or finger-ends, and in lesions of the mitral valve these signs may persist for a long time without other indications of failing compensation. In young persons this long-continued stasis may give rise to certain characteristic features. The lips become thick, the nose is broad, and the complexion dusky, while the finger-ends are bulbous and the nails incurved in the manner so characteristic of persistent congestion of the capillaries. In older persons there may be merely a dilatation of the venules of the skin of the nose and cheeks or of the extremities. In aortic disease, on the other hand, pallor and anæmia are more apt to be present, and frequently reach so extreme a degree as to merit the name of cardiac cachexia. In two recent cases in young men I found the number of red corpuscles 3,800,000 and 4,500,000, and the hæmoglobin 47 per cent. and 50 per cent. respectively. I have been particularly struck by this chlorotic condition of the blood, the hæmoglobin being diminished out of proportion to the corpuscular reduction, but have seen no cases in which the hæmoglobin fell below the figures given. Both of these were instances of aortic regurgitation; less frequently I have noted equally intense anæmia and cachectic appearance in aortic stenosis. Cyanosis may be absent in aortic affections until the mitral valve becomes insufficient, but occasionally the intraventricular pressure may sufficiently impede the circulation through the heart and lungs to lead to duskiness or venous engorgement. Sometimes in mitral cases, in addition to the cyanosis, the skin presents a somewhat jaundiced appearance. This is especially marked in the cases in which congestion of the liver and catarrh of the intestines and bile-ducts have become decided. I have also seen in a few cases a petechial eruption in the skin, doubtless dependent upon the separation of small fragments of the diseased valves or of the deposited fibrin, and their lodgment as emboli in the terminal arteries of the skin; but the symptom is one which should always lead to the most careful search for indications of acute endocarditis, particularly that of malignant type.

With increasing obstruction in the peripheral circulation dropsy finally develops. It is common in mitral disease, but rare in affections of the aortic valve, until relative insufficiency of the mitral leaflets is established. Cardiac dropsy, as a rule, begins as œdema in the feet and ascends. It is first noted at the ankles, around the malleoli, and on the top of the foot; later it extends to the subcutaneous tissues of the rest of the body, especially where the areolar structure is least compact. Extreme distention of the scrotum and penis occurs in valvular disease as in nephritis, though less commonly. The skin is usually red or cyanotic, and on careful examination dilated vessels are seen beneath the surface. At first it is soft, pitting deeply on pressure, but when œdema has been long continued a certain amount of induration of the subcutaneous tissue is developed; and, if there be excessive distention, small cracks or fissures may be produced and a scaly or eczematous condition may result. Finally serous effusion in the internal serous cavities supervenes, hydrothorax, ascites, or hydropericardium resulting. These may develop coincidently with the subcutaneous dropsy, or may be present when the latter is but slight. Liquid effusion in any of the serous cavities without external œdema should, however, always lead to the most searching examination for local causes before it is admitted to be the result of the valvular disease alone. In case of ascites the existence of cirrhosis of the liver must first be carefully excluded, and in pleural effusion the liability to a low-grade pleurisy as a complication must be considered. Yet undoubted cases of cardiac dropsy of the serous sacs without œdema of the areolar tissues do occur, and at the present writing I have under my care three cases in which hydrothorax with but moderate œdema of the ankles seems to be wholly dependent on valvular lesions.

Some of the most important symptoms of valvular disease result from the venous congestion of the internal organs, particularly the lungs, the liver, and the kidneys.

The influence of congestion of the pulmonary circulation in producing dyspnœa and other disturbances of the respiration has already been alluded to. Associated with the dyspnœa there may be cough and expectoration. The cough is frequently of a peculiar harassing nature, and may prevent the sleep of the patient. In cases in which the circulation is persistently impeded there is great liability to bronchial catarrh, a complication which lends additional severity to the existing symptoms. The expectoration is usually watery, and may be quite copious. Particular attention has been called to the presence in the sputa of certain pigment-bearing cells. The latter are either epithelial cells of the terminal bronchioles or leucocytes which have taken up the pigment matters derived from the extravasated blood; and by their numbers the degree of pulmonary congestion may be roughly estimated. When the latter is excessive the expectoration becomes blood-tinged or actual hæmoptysis supervenes, especially in stenosis of the mitral orifice. I have observed more than one case of mitral stenosis in which there were repeated hæmorrhages from the lungs, the quantity of blood at each hæmorrhage being remarkably large. After each hæmorrhage the patient's general condition was greatly improved by the lessening of pulmonary congestion. Pulmonary embolism gives rise to sudden and decided symptoms. The patient may be seized with violent shivering, and excessive dyspnœa is developed rapidly. There may be vague pain in the chest, and the sputa usually become blood-tinged, if not completely hæmorrhagic. On physical examination more or less extensive dulness, with diminution of the breath-sounds and liquid râles, is detected. If the bronchi are not occluded, the breathing is broncho-vesicular or even bronchial in character.

Congestion of the liver leads to enlargement of that organ, so that its lower border extends below the margin of the ribs, and may even be found on a level with the umbilicus. There is usually in the early stages a certain amount of pain, but this rarely becomes an urgent symptom. Congestion and enlargement of the liver reach the maximum degree when the tricuspid valves are insufficient, and it is in such cases that the organ is found to pulsate with each ventricular contraction. Enlargement of the left lobe is sometimes recognized by distinct resistance to palpation in the epigastrium, or there may be even a visible swelling and pulsation. The congestive changes in the liver-substance and the associated catarrh of the bile-ducts are productive of functional disturbances of the organ and of jaundice. The latter is rarely decided, but there is often a slight yellowness of the sclerotic or an icteroid hue of the skin. Long-continued obstruction of the hepatic circulation leads to cyanotic induration of the liver, and thereby to greater impediment in the portal circulation.

Congestive enlargement of the spleen is rarely marked until the later stages of valvular disease, with failing compensation, supervene. Previous to this it may be felt below the ribs, but later the enlargement may reach an extreme degree. Embolism of the spleen is, as has been seen, common in the malignant form of endocarditis, but, although much less frequent, it may occur in chronic valvular disease and be attended with the usual symptoms.

The mucous membrane of the stomach and bowels becomes congested and catarrhal, and in a majority of cases, especially of mitral disease, gastric and intestinal disturbances form an important part of the symptoms. The mucous membrane is thickened and covered with tenacious mucus, and flatulence, pyrosis, vomiting, and other symptoms of chronic gastritis are experienced. Occasional or persistent diarrhœa may mark the case, and the stools are sometimes copious and watery. Sudden pain in the abdomen, with distention and rapid development of collapse, is indicative of embolism in the stomach or intestines. In the same cases there may be violent vomiting and hæmorrhagic diarrhœa. Hæmatemesis sometimes results from rupture of an over-distended vessel.

Stasis of the renal circulation may be indicated by pain in the back, but more frequently it is the character of the urine that first arrests attention. When the failure in compensation is decided, the quantity of the urine decreases notably ; the color is dark, the specific gravity increased, and a deposit of urates—or, as Sir William Roberts has designated them, of quadriurates—results. Albumin in small quantity is often present, and a few casts and cylindroids may be found in the sediment. The congestion may be so intense that renal secretion is practically abolished, and some of the profound nervous symptoms of the last stages are evidences of uræmic intoxication. On the other hand, when the indurative changes of chronic congestion are established, an increase of the urine, with diminution of specific gravity, may be noted. The occurrence of renal embolism is rarely attended by decided symptoms, but suspicion would be aroused by the occurrence of blood in the urine or by sudden pain in the loins.

Hæmorrhages from the lungs and stomach have been referred to. There may also be hæmorrhages from other mucous surfaces. Epistaxis is especially common in stenosis of the mitral orifices, though the most obstinate and dangerous epistaxis I have observed in cardiac disease was in a case of aortic roughening with atheroma of the aorta, and doubtless with degeneration of the small vessels also. The menstrual function is apt to be disordered, and profuse bleeding may occur.

Nervous symptoms may from the first be prominent, or may only be developed toward the end of the disease. They are far more commonly present in cases of aortic lesions than in mitral, and doubtless to a large extent depend upon the coincident arterial changes, which add to the impairment of the cerebral circulation occasioned by the valvular defect. In mild cases there is often vertigo and tinnitus aurium when the patient makes any special exertion, or these symptoms may be constant as long as the patient maintains the erect posture. In other cases the patient is liable to sudden syncopal attacks, and may fall while in the streets or at his work. When the compensation fails and the cerebral circulation is continuously impaired, persistent headache is often noted. Sometimes it has the character of the hysterical clavus, and may be a most distressing symptom. Disturbance of sleep or complete sleeplessness may be associated with headache or may be an independent symptom. Even loss of memory and psychoses of various kinds may result from the disturbance of the cerebral circulation. Melancholia with suicidal tendency is most frequent, but maniacal excitement is occasionally observed. A patient of my own was noted to grow more and more despondent, and finally ended her life by swallowing crude carbolic acid. Preceding the fatal termination in any case the patient frequently becomes delirious or stuporous and comatose, and it is in these cases also that Cheyne-Stokes respiration is frequently observed. In all cases in which the nervous symptoms become pronounced the urine should be carefully examined, since uræmic poisoning probably has an important part in their production.

Cerebral disturbances may also result from embolism, and especially in aortic disease cerebral hæmorrhage is not unusual. The embolic manifestations may consist of transient disturbances of consciousness and palsy, or may present themselves in the form of decided embolic apoplexy, with permanent hemiplegia. Epileptiform convulsions have sometimes been observed, and inveterate epilepsy seems to have occasionally developed after embolism from a diseased valve. The relation of chorea with valvular disease is probably more often that of cause than of result, but it may be that some cases are due to minute cerebral embolisms.

Embolism of the peripheral vessels is a comparatively rare accident in valvular disease. When one of the larger arteries of the extremities is occluded, the limb grows cold and blue and rapidly undergoes gangrenous changes. At first there is excruciating pain, but as the mortification increases and the parts grow more black in color, complete death of the parts is indicated by the cessation of pain. Finally, septicæmia may result from absorption of septic matters from the affected tissues, and lead to the death of the patient. The left leg is more frequently involved than the right, probably because the left iliac artery is a more direct continuation of the aorta than is the right.

Smaller emboli in the vessels of the skin may produce a petechial eruption, and I have known of cases where there were larger areas of subdermal suffusion. Thrombosis of the veins of the extremities, particularly the femoral, is more frequent than embolism. It results from the slowing of the circulation, and is therefore an indication of marked disturbance of compensation. Increasing distention of the veins, with œdema and great pain, ensue, and sometimes fragments of the clot are washed into the circulation as emboli. Under similar circumstances thrombi frequently form in the appendices of the auricle or in the auricle itself. In the latter situation they may be of a curiously globular shape, and may lead to complete occlusion of the valvular orifices and to rapid death. Globular thrombi may also be found in the ventricles: I have observed them three-quarters of an inch in diameter, yellowish in color, with advanced central softening so as to be little more than a fibrinous sac filled with puriform contents, attached to the trabeculæ and in one instance with sufficient mobility permitted by the bands of attachment to allow the thrombus to partly occlude the mitral opening.

The general health of a patient suffering with a chronic valvular disease may seem remarkably good. Emaciation is rarely seen, even in the cases in which anæmia is extreme and local symptoms are severe. As chronic valvular disease in itself is usually afebrile, the occurrence of fever should always lead to a careful physical examination to determine the existence of an acute exacerbation of the valvular disease, since recurring endocarditis is so frequently observed. Especially should this occurrence be suspected in cases of irregular fever with chills and embolic symptoms. In other cases the fever may be due to intercurrent inflammation of the serous membranes, lungs, or other organs.

I have repeatedly had reason to suspect that a febrile process which did not seem referable to renewed valvulitis was due to an extension of inflammation to the intima of the aorta; and recently a case of chronic mitral disease terminated where during a course of several years there were outbreaks of repeated chills followed by high fever, subsequent to which areas of severe arteritis appeared, leading successively to aneurism of the popliteal, femoral, and axillary arteries. Embolism of the central artery of the left retina also occurred.

Complications.—Many intercurrent affections may arise in the course of chronic valvular disease which are in no sense direct or indirect results of the valvular lesion, and cannot therefore be reckoned among the complications. There are others, however, to which the system is predisposed by the cardiac defect or which follow as immediate consequences.

The diseased valve must, in the first place, be looked upon as distinctly liable to renewed attacks of acute endocarditis, especially that of malignant type, and the occurrence of fever should lead to the suspicion of such a complication. In a case of aortic regurgitation in a young man recently under my care the temperature was found to be rising slightly in the evening without other symptoms. The pulse and respiration were somewhat more rapid, but the physical signs remained unaltered, and no positive evidence of the nature of the fever was obtained until three months after the onset, when splenic and renal embolism determined the diagnosis. Soon after this the patient died, and at autopsy advanced ulcerative endocarditis, affecting the aortic and mitral leaflets, was discovered.

Pericarditis may complicate the valvular disease at any stage of its course. It more frequently occurs in aortic than in mitral disease, extension of the inflammation taking place more readily there on account of the thinness of the wall of the aorta. Pericarditis may also be associated with endocarditis at the very onset of the latter, the two diseases arising from a common cause. This is particularly true in the case of children and young persons.

The intimate relations of valvular disease with general arterial sclerosis have been alluded to. The same sclerotic process may also invade the kidneys, the spleen, and other organs. Of particular interest is the association of renal, vascular, and cardiac sclerosis, the whole process resulting from some general toxic influence—alcoholic, gouty, syphilitic, or the like. In these cases there is more or less involvement of the vessels in all parts of the body, causing thickening of their walls and increased arterial tension; the kidney is cirrhotic and contracted; and the heart, in addition to the valvular lesion, is invaded by the same sclerotic process, which leads to thickening of the fibrous tissue between the muscle-fibres, and finally to degenerative changes of the latter from pressure and diminished nutrition. The same arterial, renal, and myocardial processes may, however, be present without a valvular lesion. Clinically, cases of this kind are recognized by the high arterial tension, the copious light-colored urine, which may contain a small quantity of albumin and hyaline casts, and by the enlargement of the left ventricle. The dominating condition, as Huchard has well said, is the general condition, and not the valvular lesion.

Nephritis, however, of the ordinary type may arise at any stage of valvular disease, and is a grave complication. Uræmia may occur as a result of such intercurrent nephritis or from mere stasis of the renal circulation, without decided anatomical changes. The marked nervous symptoms of the later periods may sometimes be attributable to this cause.

Pleurisy and pneumonia are not unusual complications. In particular, is pneumonia of hypostatic type a common and grave condition. Œdema of the lungs is perhaps the most frequent cause of death, and chronic bronchitis, though less serious, is often a most troublesome complication.

The embolic complications have been sufficiently described under Symptomatology. Occasionally, when the skin of the extremities becomes fissured by the excessive œdematous swelling, erysipelatous inflammation results, and may lead to rapid septic infection and a fatal issue.

The disordered state of the circulation leads to constant congestion of the mucous membranes of the stomach and intestines, which strongly predisposes to catarrhal inflammations. Undue exposure or indiscretions in diet are therefore often productive of serious gastritis or enteritis, and in many cases, by their repetition, chronic catarrh of the stomach and bowels becomes established.

Diagnosis.—In few, if any, diseases can it be said with greater truthfulness that the determination of the name of the disease has not at all completed the diagnosis. It will be necessary to distinguish valvular disease from other conditions simulating it, to determine the seat, the nature, and the degree of the valvular defect, and to estimate the effect of the compensatory changes as well as the secondary alterations in other parts of the body, before the diagnosis may be said to be complete.

The recognition of valvular disease rarely presents much difficulty if careful auscultation be practised. Sometimes the cardiac symptoms are so obscure that attention may not be directed to the heart. This may occur in cases in which nervous symptoms take a prominent place, but a careful physical examination should never be neglected in obscure diseases of every kind, and rarely fails to discover the valvular lesion. When doubt exists, repeated examinations must be made in varying conditions of tranquil or excited cardiac action, in different states of respiration, and in different positions of the patient's body.

When a patient presents universal œdema, it will be necessary to distinguish between cardiac and renal dropsy, and the diagnosis is rendered somewhat difficult by the fact that albumin and casts are frequently found in the urine of purely cardiac cases, and that a cardiac murmur may coexist with nephritis. It is to be noted, however, that in the dropsy of Bright's disease puffiness is apt to be first observed in the loose tissue of the lower eyelid, whereas in valvular disease the ankles and feet are first affected, and that, while there is usually marked pallor of the skin in renal dropsy, there are cyanosis and distention of the venules in valvular disease. Moreover, in the latter the external genitals are neither so commonly nor so early affected as in Bright's disease. Finally, the character of the casts in the urine and careful examination of the pulse and action of the heart will serve to make the distinction between the two affections, or in the case of their coexistence will determine which is the more serious condition in the case.

Effusion in the serous cavities unassociated with external œdema are rarely met with in valvular disease, and should always lead to a searching examination for local causes. In case of pleural effusions inflammation of the pleura may be discovered by the existence of fever or a history of pain in the side, or by the character of the liquid aspirated; but occasionally a pleural exudation may come on insidiously without the slightest fever or local evidences of inflammation. In the latter case the absence of cardiac murmurs is most significant.

Ascites is much more frequently due to cirrhosis of the liver, and even when valvular disease is present the occurrence of ascites without external œdema must be considered as suggestive of cirrhosis.

The distinctions between cardiac disease and aneurism of the aorta will be more appropriately considered in the discussion of the latter condition, as will also the diagnosis of simple hypertrophy and dilatation of the heart after the description of these affections.

Patients with chlorosis and extreme anæmia frequently present themselves with marked palpitation of the heart as the sole or prominent symptom. In these cases a diagnosis of cardiac disease is frequently made on account of the existence of murmurs over the heart, and it may be most difficult to distinguish such anæmic murmurs from those of organic valvular disease. It must also be remembered that swelling of the feet or universal dropsy may result from an anæmic state of the blood. Attention has been called to the fact that, especially in aortic disease, the appearance of the patient may be highly anæmic or cachectic, and that a low blood-count and percentage of hæmoglobin may be found. The anæmic bruits are, however, generally soft, and are always systolic. They are heard with maximum intensity at the base of the heart or in the pulmonic region. At the same time there is often heard a peculiar musical humming sound, the bruit du diable, or venous murmur, in the jugulars. The murmurs of valvular disease are recognized by their greater harshness and their transmission in definite directions. Diastolic murmurs are never due to anæmia, and the existence of hypertrophy of the heart or characteristic changes in the pulse would definitely establish the diagnosis.

Patients are frequently startled by discovering irregularity of the pulse or by palpitation or pain in the præcordial region, and come to the physician with the fear that they have incurable valvular disease. Such symptoms do indeed occur in cardiac disease, but it is fortunate that we can assert that they are far more commonly functional in nature, resulting from a variety of trivial conditions. Careful physical examination will then show an absence of valvular lesion, and further study discloses the digestive disorder or other local affection which has by reflex irritation excited the functional disturbance of the heart.

The particular form of valvular lesion present in a given case will be determined by applying the methods of physical examination, and particularly by noting the nature of the cardiac hypertrophy, whether of the right or left ventricle, and by studying the time and distribution of the murmur. The seriousness of the valvular defect is mainly to be measured by the effect upon the circulation and the size of the heart, and not by the loudness of the murmur. It is not unusual to find very loud murmurs when there is but a slight roughening of one of the valves, while a scarcely audible one may attend an advanced lesion.

The symptoms give important indications of the exact pathological conditions present, especially when the coexistence of two or more murmurs makes it necessary to determine which lesion is mainly responsible for the general condition of the patient. Distention of the venous system, with congestion of the viscera and with dropsy, is more frequently indicative of mitral than of aortic disease; and when marked cyanosis and extreme dilatation of the veins are observed, failure of the tricuspid valves may generally be inferred with reasonable certainty. Anginoid pain, anæmia, and vascular sclerosis, on the other hand, would indicate a special implication of the aortic valves, as would also the occurrence, in the early stages, of syncopal attacks, vertigo, and other nervous manifestations of improper cerebral circulation.

The distinction of rheumatic from sclerotic—or, as they have been called, cardio-sclerotic—cases of aortic disease is not as readily made as some authors, particularly the French writers, maintain. As a rule, however, in young subjects, in whom arterial changes are slight and the pulses bounding, a rheumatic history will be obtained, and the anatomical changes will be found to affect principally the valves, whereas in persons past middle life, with rigid arteries and less bounding pulses, syphilis, alcoholism, or gout may be determined, and the pathological process will be diffuse, affecting the whole arterial system.

Prognosis.—The prognosis in valvular disease must be based upon a careful study of the nature of the disease and the degree of compensation present, as well as upon the general health of the patient, his occupation, and his habits. The degree of compensation depends to a large extent upon the health of the patient. In cachectic or neurasthenic persons an extreme valvular defect may call forth but little compensatory hypertrophy, and the prognosis of course is correspondingly grave. After the establishment of complete compensation the prospect of its continuation will be largely influenced by the patient's occupation and habits. If he be required to continue at active or laborious work and be exposed to vicissitudes of weather, the liability of early failure of the cardiac power is very great. Similarly, indiscretion in diet, the immoderate use of alcoholic stimulants or of tobacco, or undue mental excitement will constantly tax the strength of the heart, and eventually aid in destroying compensation. It is by no means implied that moderate, healthful exercise of body and mind are to be looked upon as dangerous, for many patients undoubtedly are thereby benefited, and are enabled to continue long and useful lives in spite of a serious defect of one of the valves of the heart.

Intercurrent diseases of all kinds render the prognosis grave. The rapid supervention of cardiac degeneration and dilatation in the course of severe infectious fevers has already been referred to, and suggests the adoption of special precautions in persons suffering with valvular disease. Pulmonary diseases of all kinds directly tax the power of the right ventricle, and are far more serious in persons with valvular lesions than in healthy individuals.

Compensation is more completely established and more permanent in aortic and mitral regurgitation than in other lesions, but life is often prolonged for many years in case of aortic and mitral stenosis in spite of the fact that certain symptoms of incomplete compensation had been present from the beginning. Tricuspid regurgitation is generally itself the result of beginning failure of compensation in a case of mitral disease, and therefore the prognosis is particularly grave. Tricuspid stenosis, on the other hand, may permit comparatively comfortable life for many years. Among the 46 cases tabulated by Fenwick, the duration of life was from thirty-one to thirty-six years.

Aortic regurgitation, though highly favorable in other respects, is more liable than any other form of valvular disease to terminate in sudden death. On the whole, however, in striking contrast with the popular notion on this point, sudden death is a rare accident in valvular disease. Most cases terminate slowly from gradual failure of cardiac power, with increasing dropsy and numerous internal engorgements. There are usually, therefore, decided indications of the approach of the fatal termination. Among these may be specially named excessive dropsy, great irregularity of the heart's action and weakness of the pulse, increasing failure of the renal secretion with abundant albuminuria, and the development of profound nervous symptoms. Cheyne-Stokes respiration in heart disease nearly always presages approaching death.

Destruction of a valve by traumatism usually leads at once to extreme dyspnœa and cyanosis and to a fatal termination, but sometimes a certain degree of compensation is established, and life has been prolonged as much as five years in some recorded cases.

The possibility of a valvular lesion resulting from chronic endocarditis undergoing complete resolution has been much discussed. There is, however, no proof that such a thickened valve ever regains its natural flexibility, though competent observers have claimed that physical signs of undoubted organic disease have disappeared under their observation. I have already expressed the opinion, based on cases observed closely for a number of years, that acute endocarditis, especially in the young, may terminate in complete recovery, with the gradual disappearance of a decided murmur. This is, however, a widely different matter from the cure of a chronic lesion, and it may be questioned whether the murmurs in the cases recorded in support of such an occurrence may not have been due to mere relative failure of the valve without actual organic disease.

Treatment.—The treatment of chronic valvular disease must be varied to suit the stage of the affection and the exact conditions present. It must always be remembered that no medication or other treatment at our disposal will remove the valvular lesion, and that all of our therapeutic endeavors should be directed to the establishment and maintenance of compensatory power on the part of the heart. If compensation be perfect, the patient requires rigid hygienic care, but no medicinal treatment, and the too frequent practice of prescribing digitalis or other cardiac stimulants merely because a murmur is discovered must therefore be condemned. Above all, it is the patient who must be studied and treated, and not the murmurs.

Prophylaxis.—Many cases of valvular disease following acute rheumatism might doubtless be avoided were proper hygienic care always taken in the management of the latter disease. The statistics of Sibson have shown beyond doubt that absolute rest exerts a powerful influence in preventing both pericarditis and endocarditis in rheumatic fever. The patient should from the beginning to the end of the attack be kept confined to bed and well protected from cold or drafts. Drugs have no positive power to prevent the occurrence of endocarditis, but the salicylates, by shortening the duration and by quieting the heart through relief of the pain, indirectly lessen the frequency of valvular disease. It is enough to refer to the urgent importance of closely watching for the occurrence of acute endocarditis, so that active treatment may be instituted at once. This gives the only chance of avoiding subsequent organic valvular disease or at least of lessening its extent.

In another class of cases the avoidance of muscular strain and the regulation of the diet and habits of life would reduce the proportion of valvular disease, but these are manifestly conditions over which physicians have but little permanent control. We may occasionally be consulted in regard to the education and subsequent occupation of a young boy or girl, and will do well to consider hereditary tendency to cardiac disease in giving our advice; but too often it is only after full development of the disease, or still later, when compensation is beginning to weaken, that we are called upon to treat the case.

During the stage of compensation it will first of all be necessary to direct the patient in every detail of his daily life. This requires tact on the part of the physician, for it may be desirable to conceal from the patient himself the nature of his disease. On the other hand, there are many whose earnest co-operation in treatment can only be secured by a frank explanation of their condition, and who are not injured in the least by a knowledge of the existence of organic disease. The determination of the proper course to pursue will depend upon the individual character of the patient and upon surrounding conditions. Since we may honestly dispel the dread of sudden death, and can often promise such good and lasting results if compensation be established, and since this result is scarcely possible without the intelligent co-operation of the patient, it seems proper in most cases to disclose the fact of organic disease, but in such an encouraging manner as will rouse energy and caution instead of favoring depression or recklessness.

The work of the patient should be easy. He should not become fatigued excessively, and at no time must he make violent exertion for even the briefest time. Absolute cessation of labor is not usually desirable; on the contrary, moderate exercise will prove beneficial in developing the compensation and in preserving that which has been gained. Oertel has recently instituted a plan of treatment for developing the heart's power by graduated exercise. The patient is directed to ascend inclined planes of increasing steepness and length until the compensation is fully developed. The treatment is carried out in great detail at various special resorts, but it is by no means applicable to all cases, and when desired it can practically be as well accomplished at the patient's home. Use may be made of neighboring acclivities or even of the stairs of the house. It is always necessary, however, that careful supervision should be exercised, lest undue exertion be made and the cardiac power be injuriously taxed. The tolerance of individual patients of this plan of treatment, or of exercise in general, will be found to vary widely. It is not rare to find a person dying at an advanced age with a valvular lesion of many years' duration, whose life had been one of constant physical and mental strain. A recent case of this kind impressed itself strongly upon my mind. A man of sixty-eight years, who lived in a rather mountainous district, and had all his life been an active hunter and horseman, was unconscious of any illness or physical defect until eight weeks before he consulted me. I discovered decided evidences of narrowing and regurgitation at the aortic valve, and the patient died within a few weeks. At the autopsy lesions of very long standing were discovered, consisting of the most extreme narrowing and calcification at the aortic valve, great enlargement of the left ventricle, and advanced fatty degeneration of the muscle. On the other hand, there are many patients who with but a slight valvular defect are unable to make the least physical exertion without serious disturbance of the heart's action. No fixed rule can therefore be laid down regarding the amount of work which a patient may or may not undertake, but each case must be carefully managed according to the indications present.

Mental exertion and mental excitement exercise a powerful influence on the heart, and must be restricted to the minimum. Perfect calm should always be insisted upon. In the case of children with organic heart disease this part of the treatment gives extreme anxiety. They often seem especially restless and difficult to restrain. Fatigue or exposure favors recurrences of rheumatism and acute endocarditis. Undue restraint tends to render them nervous, and even more sensitive to atmospheric changes. Play and exercise must be carefully and patiently graduated: study cannot be neglected, but must be cautiously adapted to the case. Especial care must be taken to avoid fright or sudden emotions of any kind. Older persons should so arrange their work and habits that the greatest serenity of mind may at all times be preserved. Hirsh pointed out the great frequency of valvular disease during and after the troublous times of the French Revolution, and again in 1830 and 1848, and attached importance to mental excitement as a cause of heart disease. It is more probable, however, that latent cases, and such as would remain latent under ordinary circumstances, were called into prominence by the stirring events of those anxious years. The frequency with which severe functional disorders of the heart were noted in America for some years after 1863 will be remembered by many.

The diet of the patient will always require special regulation. The quantity of food taken at one time must be small, because the distention of the stomach incident upon a full meal will directly interfere with the heart by pressure, and often disturb its action markedly. Carbohydrates and fats are badly borne, since they tend to decomposition and to gaseous distention of the stomach. Simple food taken in small quantities will always prove the best. On the other hand, coarse foods or such as tend to ferment are immediately unpleasant, and, further, are apt to lead to gastro-intestinal catarrh, to which the congested condition of the mucous membranes makes the patient unusually liable. For the same reason alcohol as a beverage must be excluded, though a small quantity of light wine or of much-diluted spirit may be permitted as an aid to digestion in persons who have been accustomed to its moderate use, and especially in patients of advancing years. Taken excessively, there is great danger of gastric irritation and of serious disturbance of the heart itself. Tobacco should not be used in any form, and tea or coffee only in small quantity and very weak. Excessive use of tea and coffee is a frequent cause of functional disturbance of the heart, and in valvular disease leads to unpleasant symptoms.

A morning sponge-bath, followed by gentle friction of the skin, is a powerful aid to the general nutrition and renders the patient less liable to attacks of bronchitis. Warm salt baths and such as are rich in carbonic acid have been highly recommended, but are not well borne in many cases. Hot baths or vapor-baths must not be permitted.

The clothing of the patient should be regulated most carefully to the climatic conditions. Woollen underwear is the best during both winter and summer, the weight being adapted to the climate and season. Unnecessary clothing renders the patient more liable to colds, and must therefore be avoided.

Constipation can usually be avoided by proper diet and regimen. If any laxative be required, it should be a mild saline or one of the natural mineral waters, such as Friedrichshall or Hunyadi Jânos. A half teaspoonful or teaspoonful of Carlsbad salt in six or eight ounces of hot water, taken before breakfast, is gently laxative and has a beneficial effect on the gastric and intestinal mucous membranes. In obstinate cases more vigorous means may be needed, but powerful purgation should never be permitted. In cases where there is evidence of hepatic congestion and torpor an occasional small dose of blue pill may be of special service. Attention must be paid to the general health of the patient when the nutrition is poor and anæmia is present. Tonic doses of quinine or the simple bitters are frequently of service, and I have found the following a happy combination :

℞. Quininæ sulphatis,		gr. xxiv vel xlviij ;
Acid. muriat. dil.,		fʒij vel fʒiij ;
Tr. gentianæ comp.,		f℥iij ;
Tr. cardamomi comp.,	q. s. ad	f℥vj.

M. et filtra.

Sig. One teaspoonful in water after meals.

When the patient is anæmic iron or arsenic may be given in addition, and cod-liver oil may be of advantage when the patient's general nutrition is impaired.

Much has been written regarding excessive hypertrophy and its treatment, but it may be questioned whether such a condition is actually of frequent occurrence. If, however, constant throbbing of the vessels and the consciousness of the cardiac impulse render the patient uncomfortable, sedatives may be necessary, but should always be given with care, lest the heart be unnecessarily depressed. Two-drop doses of the tincture of aconite or of veratrum viride may be given in such cases, or nerve-sedatives, like bromide of potash or the elixir of the valerianate of ammonium, may be used. It is also advisable in these cases to pay especial attention to the condition of the bowels. The same remedies will be applicable when fulness of the head or tinnitus aurium is present.

When compensation begins to fail, the patient should at once be placed at complete rest. Rest alone may be sufficient in mild cases to restore the cardiac power. If the patient cannot lie flat in bed, a few additional pillows may serve to make him easy, or a bed-rest may be readily improvised. Perfect quiet must be enjoined and the dietetic regulations rigidly enforced.

Remedies will now be needed to stimulate the action of the heart, and of these digitalis easily takes the lead in its certainty of action and in its power. The indications for the use of digitalis are weakness and irregularity of the heart's action, with signs of failing circulation. When these indications are present in any case of valvular disease, the drug may be given without regard to the particular valvular lesion. Objection has been made to the use of digitalis in aortic regurgitation, on the ground that the prolongation of diastole produced by the remedy tends to increase the regurgitation and leads to further dilatation of the heart. As a matter of clinical experience, however, digitalis is of value in all forms of valvular disease, and should always be used when the indications of failing compensation are presented. The beneficial effects are perhaps more strikingly illustrated in mitral than in aortic disease, but the explanation of this, I am disposed to think, may be found in the fact that failure of compensation is more gradual and attended with less degeneration of the myocardium in the former than in the latter. It may be given in the form of the powder, the tincture, or the infusion. I have found no decided advantage of one of these over another, but have used the tincture more commonly than the infusion or powder because of the convenience of its administration in varying doses. Unless symptoms are urgent, it is always best to begin with small doses, and to increase them gradually or rapidly according to the necessities of the case. To adults from five to ten minims may be given at a dose, and repeated from three to five times daily; but it is often desirable to exceed this quantity, and certain patients will bear half-drachm doses for a long time without unpleasant effect. Two drachms to half an ounce of the infusion, or from one-half a grain to two grains of the leaves may be used instead of the tincture. Ordinarily, the remedy is given by the mouth, but sometimes, when the condition of the stomach forbids this mode of administration, rectal enemata of the infusion may be employed. They are apt to prove irritating, however, and are only occasionally advisable. The same objection may be urged to the hypodermic administration, though it is not of sufficient weight to prevent this mode of administration in cases of special urgency. The pain caused is often acute, and the local inflammatory reaction decided, but I have never seen an abscess produced. The glucoside digitaline is of variable composition, and has not in my hands produced the effects of the crude drug with any great regularity. It does, however, in a measure, act as a cardiac stimulant, and may be used in granules or hypodermically in doses of about a fiftieth of a grain.

Under the influence of digitalis the heart becomes slowed and steadied in its action, the force of the systole and pulse becomes increased, and the blood-pressure is decidedly elevated. The promptness with which the effects are produced and the signs of failure of compensation are dissipated is often most gratifying. When dyspnœa and œdema are present, there is soon noted a decrease in the rate and oppression of the respiration, cough is lessened, and the dropsy begins to subside. A valuable evidence of the good effect of the remedy is seen in the increase in the quantity of the urine, which loses its high color and becomes clear and lower in specific gravity. In all cases the characters and the quantity of the urine should be carefully noted as a reliable indication of the action of the drug. Sometimes it will be found in cases marked by great irregularity of the heart's action that digitalis alone is without effect, whereas a combination with tincture of belledonna or tincture of nux vomica proves beneficial.

The use of digitalis is, however, attended with injurious effects in certain cases. Not rarely it will be found difficult to administer the remedy on account of gastric irritability and vomiting. The powder is especially irritating, and may give rise to vomiting after each administration. In these cases the infusion is sometimes better borne, but the drug may be rejected when given in any form.

Regarding the so-called cumulative action of digitalis, little risk is to be apprehended if proper care be observed during the administration of the drug. Should the pulse become extremely tense and slowed below the normal, and the excretion of urine continue deficient or even diminish, the dose of digitalis should be reduced or the remedy be suspended. If full doses are required during a considerable period of time, it is judicious to reduce the amount administered for a few days at intervals of two weeks. Certainly also if the desired effect on the heart is secured, the dose should be promptly reduced, and continued at the lowest point adequate to maintain the effect. In rare instances alarming symptoms of gastric and cardiac disorder appear suddenly during the administration of digitalis. These consist of nausea and repeated vomiting, renewed rapidity and irregularity of the heart's action, and rapid or weak pulse so that collapse may be threatened. Whether these symptoms are toxic and due to a cumulative action of digitalis, or, as it has sometimes seemed to me, proceed from severe gastric catarrh, induced by digitalis or otherwise, the drug must be instantly withdrawn. Counter-irritation and the internal use of aromatic spirit of ammonia and brandy will be followed by subsidence of the alarming symptoms.

Even when it does not disagree, digitalis often proves unavailing, and other cardiac stimulants may be substituted. Caffeine will sometimes be found an admirable remedy in doses of five to seven grains. It is easily borne by the stomach, and is decidedly diuretic, but has the disadvantage of causing wakefulness in some cases. The citrate is more soluble than the alkaloid itself, and is the salt in general use. I have sometimes found the hypodermic use of this remedy of value in urgent cases, when time or the condition of the stomach forbade the customary mode of administration. It is rendered sufficiently soluble by combination with the salicylate or benzoate of sodium, according to the following formulæ of Tanret:

℞. Sodii salicylatis,	gr. xlviij ;
Caffeinæ,	gr. lxij ;
Aquæ destillat.,	f℥iss.

Each fifteen minims contain six grains of caffeine.

℞. Sodium benzoat.,	gr. xlv ;
Caffeinæ,	gr. xxxviij ;
Aquæ dest.,	fʒiss.

Each fifteen minims contain four grains of caffeine. The solutions should always be freshly prepared.

Strophanthus in the form of the tincture, of which three to eight minims may be given every three or four hours, sometimes proves valuable, but more often is unreliable. The action of strophanthus is similar to that of digitalis, and it rarely influences cases in which the latter has failed. Frequently, however, it may be useful when it is desirable to interrupt the use of digitalis temporarily. The sulphate of sparteine is a powerful cardiac stimulant and an efficient diuretic. I have found it serviceable in cases of valvular disease in which dropsy was pronounced. It may be given in doses of a sixth to a quarter of a grain in simple watery solution. Adonidin and convallaria majalis have been lauded as remedies equalling digitalis, but their reliability may be seriously questioned.

One other cardiac stimulant of undoubted value remains to be considered—namely, nitro-glycerin. It is invaluable in cases in which vascular tension is high, and therefore is more often useful in aortic than in mitral lesions. One drop of the officinal 1 per cent. solution may be given every three or four hours by the mouth or hypodermically, and the dose may be gradually increased, unless headache be produced. Under the action of nitro-glycerin the peripheral blood-vessels are dilated and the blood-pressure diminished, while at the same time the cardiac power is increased. The removal of the obstruction to the circulation in the arterioles manifests itself by the relief of the cardiac distress or pain experienced in most of the cases to which this drug is specially applicable, and by the general improvement of the patient.

In addition to the cardiac stimulants it is often essential to administer remedies whose action is that of general tonics, such as strychnine and quinine. Nux vomica, or strychnine in particular, is of great value as a general tonic as well as a cardiac stimulant. The combination—

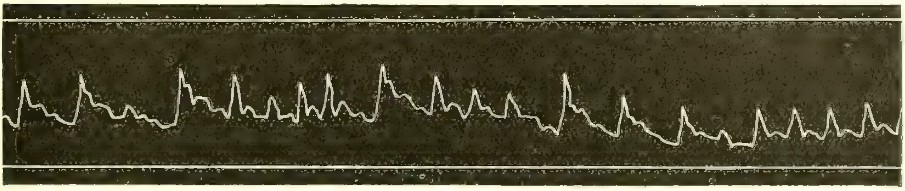
℞. Tinct. digitalis,	
Tinct. nucis vomicæ,	*āā.* f℥j,

in doses of ten or fifteen minims, may be active when digitalis alone has failed ; and especially in cases in which cardiac degeneration is advanced is its use to be commended. Strychnine alone may be given as a general tonic when compensation has been largely restored, or in larger dose I have found it valuable, given hypodermically, in sudden failure of the cardiac power. Quinine is advantageously combined with digitalis in pill form for use in cases in which the compensation is below the requirements of healthy circulation and the systemic tone deficient.

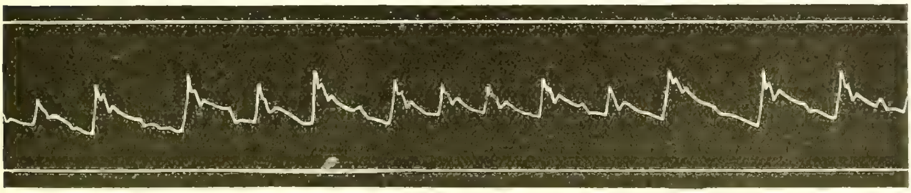
When in the course of valvular disease the heart-power suddenly fails and the symptoms become urgent, remedies are required whose action is more immediate than those which have been named. Among these whiskey or brandy, ether, ammonium, strychnine, or atropine, used hypodermically, or nitrite of amyl by inhalation, may be employed. Their action, however, excepting in the case of strychnine and atropine, is fugacious, and it is necessary, therefore, at the same time to administer digitalis or other remedies whose effect, though longer delayed, is more lasting or permanent.

In some cases of this kind, however, the entire venous system is overfilled with blood, cyanosis reaches an extreme degree, and the cavities of the heart, particularly the right ventricle, are so distended that no amount of stimulation will restore the cardiac action unless the intracardial pressure be relieved. Before I ventured to bleed in such conditions I observed several remarkable cases where, after spontaneous hæmorrhage from the nostrils or the bronchial tubes, prompt and progressive relief followed; and experience soon convinced me that in cases of this kind there can be no question of the value of venesection. A few ounces of blood taken directly from a vein of the arm relieves the heart and restores its power of contraction. In milder cases the use of leeches or the wet cup about the chest and neck or in the hepatic region may be sufficient; but when cyanosis is marked immediate venesection should be performed. In an extreme case I punctured the right ventricle itself, with instant relief of the patient, followed by permanent restoration under the use of cardiac stimulants. I cannot say, however, that further experience with this delicate procedure has impressed me with any belief in its reliability. It must at best be viewed as an extreme and almost desperate measure. It is a mistake to say that bleeding is permissible only in cases in which the pulse is strong, for in the instances in which it proves of greatest service the left ventricle is so empty of blood that its contractions are ineffectual. As soon as the right heart is

FIG. 17.



Before Bleeding.



After Bleeding.

relieved the circulation through the lungs is re-established, the left heart supplied with blood, and the pulse gains in force and regularity. The graphic illustration of these facts is seen in the preceding sphygmographic tracings from a case of mitral regurgitation. The patient was cyanosed, the peripheral veins were distended, dyspnœa was extreme. After removal of sixteen ounces of blood the pulse became slower and more regular, cyanosis disappeared, and the patient was able to lie in bed.

Bleeding is not, however, to be performed in all cases in which cyanosis is present. There are certain disadvantages which properly restrict its use to those cases in which the sudden and extreme distention of the venous system and right heart demands instant relief. After venesection the blood-vessels soon refill with serum from the tissues, and the blood is diluted to a degree which may prove injurious to the cardiac and general nutrition. This danger, however, may be trifling compared with that which threatens life, and in such cases venesection is demanded.

Relief may also be afforded the venous repletion by purgation. Mild salines may suffice, or it may be necessary to resort to more active purges, such as calomel or blue-mass, but irritating, drastic purges should be avoided on account of their tendency to induce catarrhal inflammation of the mucous membranes and to render the stomach unretentive. The treatment by administering concentrated solutions of Epsom salt or other salines, before breakfast in particular, has the disadvantage that the stomach may not tolerate the doses, and afterward prove rebellious to food or other medicines. The well-known pill of digitalis, blue-mass, and squill, with or without quinine or strychnine, is valuable in cases in which purgation seems desirable to relieve the venous system, but is especially useful in the treatment of cardiac dropsy. I have often continued such a combination, using at the same time an antiseptic mouth-wash to lessen the risk of salivation for considerable periods of time with happy effect, although no purgation followed. In one instance, owing to a misunderstanding of the directions, a lad of fifteen years, suffering with bad mitral disease and extensive dropsy which had proved rebellious to treatment, took nightly for a number of months a pill containing blue-mass in the above combination; no salivation and no purgation occurred, but a gradual return ensued to an astonishing degree of good general health. The toleration of small doses of mercurials shown by cardiac cases has often surprised me.

In the course of valvular disease special symptoms will occasionally arise and assume a degree of prominence requiring treatment additional to that of the general condition.

Dyspnœa is a prominent indication of failing compensation, and may for a long time be the only symptom. The cardiac stimulants, and particularly digitalis and nitro-glycerin, may alone suffice to relieve the patient, or purely symptomatic remedies may be required in addition. Bromide of potassium or opium may be used in these cases. Opium, in particular, is a valuable adjuvant in the treatment of cases in which subjective symptoms, such as dyspnœa and pain, are marked, but it should be used reluctantly and only in moderate doses. The following combination is valuable in cases of this kind:

℞. Quininæ sulphatis,	gr. xxiv;
Pulv. digitalis,	gr. xij vel gr. xxiv;
Ext. opii,	gr. j vel gr. ij.—M.

Div. in pil. No. xxiv.
Sig. One pill three times daily.

In desperate cases, however, the distress is so extreme that morphine is demanded by hypodermic injection. Occasionally, where it is resorted to merely to secure euthanasia, it happens that the gain of nervous tone following relief of suffering is so great as to lead to marked improvement in the cardiac action. The bromide of potassium may be given in doses of ten to twenty grains in simple elixir just before retiring, and often obviates unpleasant attacks of nocturnal dyspnœa.

When marked dyspnœa or orthopnœa is observed, a careful physical examination of the chest should be made to determine whether pulmonary œdema or hydrothorax complicates the case. If either of these conditions be detected, mild purgation may be sufficient to bring relief, but when they are marked active treatment will be required. The cardiac stimulant in use should be given freely, and in case of œdema cups placed over the back are often of service. The patient's position in bed should be frequently changed to prevent hypostatic pneumonia. In these cases also strychnine is a valuable respiratory stimulant and tonic. When hydrothorax is present paracentesis may be required. The fact that organic heart disease exists is no contraindication; on the contrary, its presence calls for the operation earlier and more often than in cases of effusion from pleurisy. In three cases, now in my wards, of cardiac hydrothorax without external œdema, repeated tapping has been performed, each time with decided relief of the symptoms.

In cases in which the sudden paroxysmal dyspnœa, called "cardiac asthma," occurs, Hoffmann's anodyne is a remedy of great value. A half-teaspoonful or a teaspoonful may be given in cold water, and repeated if necessary after one hour. Bromide of potassium given at bed-time will often prevent the occurrence of such attacks, and in very severe cases two or three drops of nitrite of amyl may be inhaled from a handkerchief.

Chronic bronchitis and emphysema, so common in elderly persons with valvular disease, are additional causes of dyspnœa, and require the special treatment adapted to those conditions.

Palpitation and other subjective sensations about the heart are frequently distressing symptoms. If over-compensation be inferred as the cause of constant palpitation, tincture of aconite or of veratrum viride may be given; but more frequently such palpitation results from nervous excitability, and is most appropriately controlled by sedatives like bromide of potassium or the elixir of valerianate of ammonium. Slight discomfort about the heart gains importance from the fact that the patient's attention is constantly fixed upon his condition. It may often be relieved by fomentations or poultices or by painting with tincture of iodine. When severe these simple measures should be supplemented by the sedative remedies just mentioned, and in bad cases morphine may be employed. When marked anginoid pains are experienced, morphine with atropine may be administered hypodermically or nitrite of amyl by inhalation. In cases of this character also it will often be found that nitro-glycerin is preferable to digitalis, the latter sometimes even increasing the cardiac distress. An ice-bag or Leiter's tubes applied to the heart or a blister over the præcordium may be of additional benefit in maintaining the effect of the sedative remedies.

Abdominal pain in association with congestion of the liver is rarely urgent, but frequently disquieting. Counter-irritation with iodine or poultices may be of value in conjunction with depletion of the portal system by saline purges. In other cases epigastric pain results from distention of the stomach or colon, and is most readily relieved by the use of carminatives and hot fomentations. Hoffmann's anodyne is of value in these cases, but chlorodyne, in doses of five or ten drops, is even more prompt and efficient.

Other gastric symptoms, such as loss of appetite, nausea, and vomiting, are frequently present. They result from the congested state of the mucous membrane, and are sometimes combined with the evidences of congestion of the liver, the bile-ducts, and the intestines. These symptoms can only be permanently relieved when compensation is restored, but may require palliative treatment. Nux vomica, gentian, or other stomachics given before the meals will often improve the appetite, and small doses of morphine, hydrocyanic acid, or chloroform may suffice to control vomiting when urgent. Nitrate of silver in pill form, combined with small doses of opium, or bismuth with creasote or carbolic acid, is of value when diarrhœa supervenes.

The most important symptom, however, in the course of valvular disease, for which special treatment is required, is dropsy. In mild cases rest in bed and moderate doses of digitalis suffice. In severe cases digitalis must be given freely. It proves effectual in dropsy by stimulating the heart and circulation, by increasing the flow of urine, and by contracting the peripheral blood-vessels. No other of the cardiac stimulants possesses all of these qualities to the same degree as digitalis, and none of them is as often of value in the removal of this symptom. Sometimes, when digitalis has failed, caffeine or sparteine or nitro-glycerin may be useful, but more commonly in such cases these remedies will fail. Remedies should at the same time be given to deplete the blood by purgation or diuresis. Calomel and blue-mass are hydragogue cathartics of great value in the dropsy of cardiac as well as renal disease. Either of them may be combined with digitalis and squill in pill form:

℞. Pulv. digitalis,	
Pulv. scillæ,	*āā.* gr. xxiv;
Hydrarg. chlor. mitis,	gr. xij vel gr. xxiv.—M.

Div. in pil. No. xxiv.
Sig. One pill every four hours.

Compound jalap powder and elaterium are also valuable purgatives, but are more irritating to the stomach and intestines. Squill is an efficient diuretic when the kidneys are healthy, but if renal irritation be present the salts of potassium are preferable, and are often combined with infusion of digitalis. Juniper in the form of gin is a powerful diuretic, but is apt to be irritating to the kidneys.

As an aid to these measures the diet of the patient should be as dry as possible, no unnecessary liquid being ingested. The use of hot-air baths or medicinal diaphoretics is to be reprobated, the cardiac depression incident to this method making it unsafe.

When the extremities are excessively swollen and the skin distended, liquid may be withdrawn by direct incision or puncture, and the embarrassment to the circulation thereby relieved; but the danger of erysipelatous inflammation is incurred by such treatment. A safer method of direct removal of fluid is the insertion of fine silver trocars with rubber tubing attached, the so-called Southey's tubes. The fluid slowly escapes through the tube, and there is little danger of serious inflammation.

In case of transudation into the serous sacs, especially those of the pleuræ and peritoneum, the ordinary treatment for dropsy may be instituted; but when this fails or serious pressure is exerted by the fluid, paracentesis must be performed and may require repetition. Attention has already been called to the conditions under which venesection may be found a valuable remedy.

Nervous symptoms, such as headache and wakefulness, require palliative treatment as long as the compensation remains impaired. Bromide of potassium, Hoffmann's anodyne, valerianate of ammonium, or opium may serve to allay the headache and to procure quiet sleep. In cases of extreme cardiac failure and suppression of the renal secretion serious symptoms of an uræmic nature may occur, and will require active treatment. Free purgation by means of the remedies before mentioned should be established, and cardiac stimulants should be given in large doses. If the latter fail on account of over-distention of the right heart, or if dangerous cerebral congestion be present, venesection affords the only hope of relief. Twelve to sixteen ounces of blood should be at once removed from a vein of the arm, and the cardiac stimulant administered in the maximum doses immediately afterward. The potassium salts may aid in promoting the flow of urine, but jalap is likely to be irritating.

Hæmorrhage from the lungs, stomach, nose, or uterus require the same treatment as in case of hæmorrhage from other causes, though active measures are rarely demanded. Cold and pressure may be applied locally if possible, and morphine should be given to quiet the patient's mind. Internal hæmostatics, like ergot and turpentine, probably have little value in the hæmorrhages of valvular disease. Styptics may be used in case of epistaxis, and in severe cases the posterior nares may require plugging. If the loss of blood has been so great as to exsanguinate the patient, hypodermic injections of warm salt water may be necessary to re-establish the circulation and prevent fatal anæmia of the brain; but the hæmorrhage is apt to be renewed as soon as the vessels begin to fill.

When the heart has regained its compensatory power the convalescence should be conducted with great care. Excessive fatigue, emotion, and hygienic errors of all kinds must be carefully avoided, and tonics may be administered with advantage, particularly such as contain iron or arsenic and strychnine. Moderate exercise should be gradually resumed, but on the first evidence of failure of compensation the patient must be put at absolute rest in bed.

Cardiac Thrombosis.

Clotting of blood within the cavities of the heart may take place immediately before or after the death of the patient, or a considerable time before the fatal issue of various diseases. In rare instances thrombosis occurs at long periods, perhaps years, before the patient's death, and the clot is found at the autopsy transformed by subsequent degenerative or formative changes. The clots which are formed in the agonal period or after death are distinguished by their reddish or yellowish appearance, by their softness, and by the fact that they are readily removed from the cavity without destruction of the endocardial lining. The color of the clot may be altered in certain diseases, as in jaundice when there is sometimes a marked icteric hue, or in leukæmia when they may be soft and of puriform appearance; and the other characters vary with the disease from which the patient suffers. True cardiac thrombi, on the other hand, are usually much harder, sometimes quite gray or white, at other times red or brown, and are firmly attached to the endocardium, so that portions of this membrane are torn off when the clot is removed.

Heart-clots are more common in the right than in the left heart, and in the auricles than in the ventricles. In the auricles they are most frequently found in the appendices, while in the ventricles the favorite seat is near the apex. They are usually more or less rounded in outline, and may be attached to the heart-wall by a broad base or by a narrow pedicle, when they resemble polypoid tumors, for which they have been frequently mistaken. These pediculated clots—or *végétations globuleuses*, as Lænnec designated them—are found most frequently near the apex of the right ventricle. Spherical clots, or "ball-thrombi," have been found in the auricles by Recklinghausen and other observers. They are more or less spherical, and entirely detached from the endocardium.

The size of the heart-clot varies from that of a pin-point to large masses which almost fill the cavity of the auricle or ventricle. Sometimes the thrombus projects through the valvular orifice, and may extend into the adjoining cavity or for long distances into the emerging vessels.

Secondary changes may take place within the clot. If life be prolonged, contraction of the thrombus occurs, and the color becomes lighter as the red corpuscles are pressed out or disintegrated. On the other hand, softening of the central portions may take place, and a somewhat cystic formation may be thus produced. Calcareous infiltration may lead to the formation of a cardiolith, such as are occasionally detected.

The **causes** which induce cardiac thrombosis are those which are operative in any case of clotting of blood within its natural channels. There may be abnormal slowing of the circulation, alterations of the blood itself, which tend to greater ease of coagulation, and roughening of the endocardium. The point at which the thrombus is attached will usually be found to present some form of localized degeneration, as, for example, an atheromatous ulcer. The frequency of such endocardial conditions, with the tendency to slowing of the circulation, accounts for the frequency of cardiac thrombosis in the course of valvular diseases of the heart. Slowing of the current may, however, be alone sufficient, as in pericarditis, myocarditis, or in various states of lowered general vitality. Changes in the blood itself, which increase its tendency to spontaneous coagulation, occur in various febrile diseases, particularly pneumonia and diphtheria, and in certain diseases not associated with fever, but in which a condensation of the blood results from loss of serum in diarrhœa or other discharges.

The **symptoms** presented in cases of cardiac thrombosis are usually indefinite in nature. Signs of cardiac weakness are generally prominent. The pulse becomes rapid and irregular, the apex-beat is weak, and the sounds may be scarcely audible. Vertigo, syncopal attacks, and dyspnœa are frequently noted, and there may be external evidences of failing circulation—cyanosis or lividity, coldness of the extremities, and the like. Embolism may occur from the breaking off of portions of the clot. Those coming from the right heart may obstruct branches of the pulmonary artery and lead to great dyspnœa, thoracic pain, cough, and bloody expectoration, with physical signs of consolidation of portions of the lung. In other cases embolic occlusion occurs in branches of the systemic circulation, especially in the spleen, the kidneys, or the brain.

Sudden death may occur when a large clot suddenly obstructs one of the orifices of the heart or one of the coronary arteries. Sudden death in the course of infectious fevers was formerly ascribed to heart-clot in all cases, but is perhaps more frequently due to other causes.

The **diagnosis** of cardiac thrombosis is practically impossible, though if great circulatory weakness be developed in the course of diseases with which clotting of the blood is apt to occur, and if embolic manifestations supervene, a probable diagnosis may be entertained.

The **prognosis** is always grave. Sudden death may occur at any time, and recovery rarely takes place.

The **treatment** must be entirely symptomatic. Cardiac stimulants must be given when the heart-power fails, but too vigorous stimulation may result in breaking off of portions of the clot and consequent embolism. Rest, suitable diet, and other hygienic measures, such as are appropriate in cardiac diseases generally, should be instituted.

DISEASES OF THE MYOCARDIUM.

BY WILLIAM PEPPER.

ATROPHY.

Definition.—Atrophy of the heart is the diminution in the size and in the amount of muscular substance of the heart, with corresponding reduction of the weight of the organ.

Etiology.—Atrophy may be general or local, and the causes vary according to the form present. Local atrophy of the left ventricle has been noted in the description of mitral stenosis, and is probably due to the decreased supply of blood passing into this cavity through the stenotic orifice. Areas of localized atrophy are also found associated with other degenerative changes of the myocardium, particularly with fibroid overgrowth. In these cases the pressure exerted upon the muscular fibres is destructive to their nutrition.

Generalized cardiac atrophy is found in old age and in many wasting diseases in which the nutrition of the entire system fails. It is rarely absent in persons who have died of long-continued cancer of the stomach, and frequently occurs in phthisis, prolonged suppuration, diabetes, and obstruction of the œsophagus. Quain found the heart decreased in size in one-half of his series of cases of pulmonary tuberculosis. More or less generalized atrophy may result from the pressure of tumors, of pericardial effusions, or of abnormal deposits of subpericardial fat; but in these cases there is often hypertrophy instead of atrophy. A moderate obstruction to the coronary circulation may be productive of atrophy of the heart from insufficiency of nutrition, while more decided coronary disease leads to myocardial degenerations.

Congenital deficiency in the size of the heart was pointed out by Rokitansky, and later by Virchow, as existing in young women suffering with chlorosis. It is generally associated with smallness of the aorta and arterial system, and with imperfect development of the genital organs. Such a congenital condition cannot be designated as atrophy, but has been appropriately named hypoplasia. In some cases it may result from arrest of development after birth. Cardiac hypoplasia is occasionally seen in men, and is not infrequent in hæmophiliacs; but in phthisis the smallness of the heart is the result rather than, as was formerly believed, the cause of the disease.

Morbid Anatomy.—The size of the heart is often decidedly diminished and its weight decreased. In one of Quain's cases, in a girl of fourteen years, the weight was but one ounce and fourteen drachms, and Bramwell records an instance in an adult in which the weight was only two ounces and twelve drachms. The surface of the heart is irregular, wrinkled, and distorted, and the coronary arteries are highly tortuous. The subpericardial fat disappears, and may be replaced by œdematous or mucus-like tissue. The muscular substance may be pale in color and quite firm ; more frequently it is dark-brown or ochre-colored and softer than normal. These cases have been called brown atrophy. Microscopically the fibres are found to be narrow, and are generally more or less granular. Sometimes fatty degeneration is quite marked, and in the cases of brown atrophy brownish pigment-granules are seen at the ends of the nucleus or filling the whole cell. They are probably derivatives of the normal coloring matter of the muscle, which is left remaining when the other elements disappear in the atrophic process.

In some cases the valve-segments are extremely thin and tenuous.

Atrophy with diminution or with enlargement of the cavities has been described under the heads concentric or eccentric atrophy respectively, but there is much doubt regarding the nature of some of the cases described. The eccentric form is doubtless often simply cardiac dilatation. The weight alone can be relied upon to decide the presence or absence of atrophy, whatever the apparent size of the heart.

Symptomatology.—The symptoms are the result of the condition inducing the cardiac atrophy, and not dependent upon the atrophy itself. The reduction in size of the heart keeps pace with the diminishing needs of the system as emaciation and anæmia proceed, and therefore symptoms of cardiac weakness are not specially prominent.

Physical Signs.—The apex-beat is generally quite weak, and may be impalpable and invisible. The cardiac dulness, both on superficial and deep percussion, is found diminished ; but the evidence obtained in this way is so unreliable, on account of the frequency of pulmonary emphysema in the old and marantic, that a positive opinion regarding the size of the heart is seldom warranted. The sounds of the heart may be weak, but are often normal, and in phthisis accentuation of the pulmonary second sound is commonly present despite the atrophy. The pulse is small and weak.

Diagnosis.—When the cardiac impulse is very weak and dulness diminished in a patient emaciated by cancer or the other causes named, the suspicion of cardiac atrophy may be entertained, but a positive diagnosis is never possible. In emphysema of the lungs the præcordial dulness may be decreased without decrease in the size of the heart, and on the other hand the presence of tubercular consolidation in the anterior portion of the left lung apparently increases the cardiac dulness, though atrophy be decided.

Prognosis.—The existence of atrophy of the heart does not affect the prognosis of the diseases with which it is associated, since the reduction in the size of the heart is proportioned to the lessened requirements of the system. The prognosis therefore depends wholly upon the underlying disease, and is not dependent upon the presence or absence of atrophy of the heart.

Treatment.—General tonic treatment, and particularly strychnine, may prevent advance of cardiac degeneration, but the treatment is mainly to be directed to the primary disease.

HYPERTROPHY AND DILATATION.

SYNONYMS.—Hypertrophia seu Hypersarcosis et Dilatatio cordis; Enlargement of the Heart; Idiopathic Hypertrophy and Dilatation of the Heart.

Definition.—The hypertrophy and dilatation of the heart which result from valvular disease have been considered in the description of valvular affections, but there is another, though smaller, group of cases in which the cardiac enlargement results from other causes, and which may be designated simply as Hypertrophy and Dilatation of the Heart, or as Idiopathic Hypertrophy and Dilatation. The term "idiopathic" used in such cases is to distinguish them from the enlargements of valvular disease, and not to imply any ignorance of the cause of the hypertrophy or dilatation; but the same name has been unfortunately applied to a group of cases in which the causation was not manifest and confusion would therefore arise from the use of this name.

The conditions, hypertrophy and dilatation, are properly considered together, because, in a great majority of cases, they are associated in the same case. Hypertrophy or dilatation may exist alone, throughout a considerable portion of a given case, but in nearly all instances the hypertrophied heart eventually dilates or hypertrophy supervenes upon an existing dilatation. Pure hypertrophy is more commonly present and is more persistent than pure dilatation.

Etiology.—The existence of cardiac hypertrophy and dilatation depends upon increased work of the heart or increased intracardial pressure, and upon the vital reaction of the muscular tissue; and the part of the heart involved will depend upon the part upon which the strain is brought to bear. Increased labor of the heart tends to the production of cardiac hypertrophy just as much as does muscular exercise to the enlargement of the voluntary muscles. Such increased labor may be purely functional in nature and due to nervous causes which excite the action of the heart, or it may be due to mechanical conditions which impede the flow of blood and would lead to its stagnation within the cavities unless relieved by hypertrophy and increase of the cardiac power. It will be seen that such mechanical impediments would first of all tend to dilate the cavities of the heart, and that the degree of dilatation resulting in a given case will depend upon the power of the heart to hypertrophy and to maintain the circulation. Two elements will then enter into the determination of the degree of hypertrophy or of dilatation: first, the nutritive condition of the heart-muscle, and second, the suddenness and severity of the mechanical obstruction. If the heart-muscle be entirely healthy and the obstruction to the circulation developed slowly, pure hypertrophy may be induced, and may persist until secondary degenerations weaken the walls and permit of dilatation. On the other hand, if impediments are offered to the circulation in a case in which high grades of myocardial degeneration are present, the heart is incapable of compensatory hypertrophy, but simply dilates before the increasing intracardial pressure. Dilatation may, however, occur even when the heart-muscle is healthy, if the strain upon the heart be unusually sudden and severe. A striking example of this, though not exactly pertinent, since it deals with a valvular lesion, is seen when one of the aortic leaflets is suddenly ruptured or detached. In this case, even though the muscle of the heart be normal, the suddenness and severity of the ventricular distention are so great that the cavity is dilated to an extreme degree and time is not given for the development of hypertrophy. Considerable distention of the normal right ventricle is seen in the case of athletes after vigorous exercise, especially running. Unless unusually taxed, the ventricle regains its normal condition when the person rests, but repeated strains of this kind may result in permanent enlargement of the cavity, and at the same time in hypertrophy of the walls if the nutrition of the muscle be good. Time, therefore, and the degree of cardiac nutrition or disease are important factors in every case, and are influenced by the individual cause of the enlargement and by the general health of the patient, the age, and the sex.

The highest grades of hypertrophy are reached in young persons, and especially in children, in whom compensatory changes of all kinds are so readily developed. It is very unusual for high grades of hypertrophy to arise in advanced life. The male sex predisposes, more by the special liability of boys and men to certain of the causes of enlargement of the heart than by any peculiar tendency to its development when such causes are present. The state of the general bodily nutrition is important in that it determines to a large extent the condition of the myocardium. The older writers assigned to plethora an important place in the etiology of hypertrophy and dilatation, and there is no doubt that excessive feeding and drinking are decided factors, whether this be due, as Niemeyer supposed, to the repeated states of plethora following each full meal or to the general repletion of high living and the consequent stimulation of the cardiac nutrition. Reference will be made to this point again, and it may be left for the present with the statement that luxurious habits of eating and drinking exert a decided influence.

With these general remarks regarding the factors concerned in the production of hypertrophy on the one hand and dilatation on the other, we may proceed to consider the causes leading to overwork and, therefore, hypertrophy of the left ventricle, of the right ventricle, and of the whole organ, and then the myocardial disturbances which predispose to dilatation under the operation of the same or similar causes.

1. The conditions which exercise their influence primarily upon the left ventricle are those which affect the general arterial circulation. Foremost among these is arterio-sclerosis, either with or without coincident cirrhosis of the kidney. The widespread degeneration and rigidity of the arteries in this condition are manifested clinically by the high degree of arterial tension, and pathologically by the great hypertrophy of the left ventricle commonly observed. The enlargement may, however, to a large extent be due to coinci-

dent overgrowth of the fibrous structure of the heart, as was well illustrated in a case of a heart at St. George's Hospital weighing forty ounces, which was looked upon as one of simple hypertrophy until Quain examined it and found the enlargement largely, if not wholly, due to fibroid degeneration. Fibroid overgrowth of the heart may, however, impede the action of the muscular tissue, and therefore induce secondary hypertrophy; and it is unusual to find marked interstitial fibrous degeneration without any evidence of hypertrophy. Hypertrophy of the left ventricle also arises in the course of subacute or chronic nephritis without arterio-sclerosis, and probably in these cases is due to the action of toxic substances in the blood constricting the arterioles and raising blood-pressure or acting directly upon the muscle of the heart.

Congenital narrowing of the aorta is not uncommonly present at the orifice of the ductus Botalli and leads to extensive hypertrophy of the left ventricle. A similar mechanism is operative in cases of intrathoracic tumors or of curvature of the spine, in which pressure upon the aorta or larger arteries in the narrow portion of the thorax impedes the circulation. The influence of aneurisms is not so certain, for very large aortic aneurisms are sometimes observed without much or any hypertrophy. If, however, the arterial dilatation be of such nature that the circulation must pass directly through it, hypertrophy can scarcely fail to occur, and sometimes reaches a high grade. During pregnancy there is often moderate hypertrophy or dilatation of the left ventricle, as Larcher first pointed out: this is doubtless due to the increased force required in the maintenance of the placental circulation and to overcome the effect of abdominal pressure. Gerhardt has questioned the accuracy of this opinion, ascribing the apparent hypertrophy to elevation of the diaphragm and consequent displacement of the heart against the chest-wall, but observers are generally disposed to accept the view of Larcher. The hypertrophy usually subsides during the puerperal period, but may be permanent after repeated pregnancies.

A group of cases has been described under the name of idiopathic hypertrophy, in which the causes are not discoverable in any anatomical conditions present at autopsy. Many of these are due to the influence of severe muscular exertion, as was pointed out by Clifford Albutt, Seitz, and others in the case of mechanics whose work is laborious, and by Myers and DaCosta in the case of soldiers. The explanation of the hypertrophy is found in the fact that the peripheral vessels are compressed and the blood-pressure elevated by the muscular contractions. There may or may not be coincident arterio-sclerosis resulting from the overwork and increasing the tendency to hypertrophy. The influence of muscular exertion is seen in the case of athletes, who, unless their exercises are very prudently supervised, are apt to exhibit some hypertrophy of the left ventricle or of the whole heart. Zielonko, and later Roy and Adami, showed this effect of increased pressure by experimental constriction of the aorta. Hand in hand with the effects of muscular exertion may be ranked immoderate use of alcoholic beverages, particularly such as are taken in large bulk, like beer. Bollinger, Strümpell, and other German writers have called particular attention to this cause, and the first-named observer noted excessive weight of the average adult heart in Münich. In these cases the nutritive influence of the beverage may contribute to the overgrowth of the heart, as well as the constant strain occasioned by the ingestion of large quantities of fluid. In the same class of cases may be reckoned those in which over-indulgence in food and drink of other kinds is operative.

2. Hypertrophy of the right ventricle may follow that of the left as a result of the increased pressure of the blood in the left ventricle, and the consequent difficulty of maintaining the pulmonary circulation against this impediment. Such secondary hypertrophy may result even when there is quite pure hypertrophy of the left ventricle, but reaches its highest grade when dilatation of this cavity renders the mitral leaflets incompetent, and the blood regurgitates into the auricle and pulmonary veins. Another explanation of the coincident enlargement of both ventricles may be found in the fact that the habitual association of their action may lead to overwork of both when strain is brought to bear upon either, and in the fact that certain muscular fibres are common to both, and therefore influenced by a common cause.

Generally, however, hypertrophy of the right ventricle is due to interference with the pulmonary circulation, as is seen in emphysema, phthisis, or cirrhosis of the lung. Pressure upon the pulmonary artery and congenital narrowing of this vessel are rare causes, as are also curvature of the spine and extensive pleuritic adhesion.

3. Hypertrophy of the auricles, unaccompanied with stenosis of the auriculo-ventricular orifices, is much more rare than that of the ventricles; and is usually secondary in importance to the coexisting dilatation. The left auricle may hypertrophy considerably when dilatation has supervened upon hypertrophy of the left ventricle and the mitral leaflets have become incompetent, or even before that point has been reached.

4. Hypertrophy of the heart as a whole may result from successive involvement of the several chambers, or may be induced by causes which occasion increased work of both sides. Among such cases are exophthalmic goitre, hysteria, or mental states which lead to constant or frequently repeated overaction of the heart. Tea, coffee, tobacco, and alcohol used immoderately probably act in the same way in certain cases. The pressure of pericardial fluid and the resistance offered by pericardial adhesions are causes of great importance, inducing hypertrophy of the whole heart, though primarily and principally that of the left ventricle. The influence of pericardial adhesions has been questioned by certain writers, who would ascribe the cardiac enlargement to coexisting valvular disease rather than to the adhesions; but hypertrophy and dilatation have been found, according to Quain, in at least a third of the cases in which no valvular disease existed. Theoretically, it is easy to conceive of hypertrophy resulting from the immoderate work imposed upon the heart by adhesive pericarditis, and clinically my experience would lead me to accept, or even to magnify, the proportion set by Quain.

Myocardial degeneration of some kind will usually be found in cases in which dilatation is in excess of hypertrophy, and may be induced by various diseases. In the course of infectious fevers parenchymatous degeneration and acute myocarditis are frequent causes of weakening of the heart-muscle, and may lead to sudden and fatal cardiac dilatation unassociated with hypertrophy. These conditions have been noted particularly in typhoid fever, diphtheria, articular rheumatism, and scarlet fever, and my clinical experience inclines me to include influenza. More gradual degeneration and yielding of the muscular substance occurs in persons addicted to immoderate use of alcoholic beverages and in those suffering general nutritional disturbances or arterial sclerosis. In these cases fatty degeneration, fatty infiltration, or fibroid overgrowth is commonly observed. The extreme dilatation sometimes met with in adherent pericardium is doubtless to a certain degree due to the opposing forces of the adhesions and the ventricular contractions, but is also in part a result of the fibroid myocarditis associated with the pericardial disease. The most extensive dilatation occurs when the parietal pericardium is adherent to the chest-wall and pleura and to the heart. Anæmia and chlorosis are not infrequent causes of myocardial degeneration and dilatation. Occasionally marked dilatation is found to be present when the microscope fails to detect cardiac degeneration of any kind. It is impossible to account for such a condition, excepting by the somewhat vague assumption that the cardiac tone is lowered in a functional manner. Perhaps the muscular innervation, of which we know so little as yet, is disturbed in these cases, but this is entirely theoretical. A few observers have described morbid alterations in the cardiac ganglia.

Hereditary influence has been asserted to predispose to dilatation, but is certainly not a marked factor. The unlikelihood of hypertrophy developing in persons of advanced age has been referred to, but dilatation, on the other hand, is apt to make manifest the cardiac weakness and degeneration common to this period of life.

Dilatation may occur primarily, coincidently with hypertrophy, or after the latter. Coexisting hypertrophy and dilatation are met with when the circulatory impediment is of such nature as to require increased cardiac power and at the same time increased size of the cavities, both conditions being therefore conservative in nature. An example of this, again not altogether pertinent, because it involves a valvular lesion, is seen in aortic regurgitation, in which the overfilling of the ventricle by the double supply of blood—one stream coming from the auricle, the other from the aorta through the incompetent valve—necessitates ventricular dilatation, while the increased work of moving an excessive quantity of blood at each systole leads to hypertrophy. Dilatation is apt to supervene upon hypertrophy because the hypertrophied muscle is particularly prone to undergo degeneration. The muscle, weakened by degeneration, becomes less and less able to expel its contents at each systole, the quantity of blood within the cavities increases, and dilatation results from the distention of the walls.

Morbid Anatomy.—The various cases of cardiac enlargement of the heart may be described as of three forms: (1) Simple hypertrophy, in which the walls are thickened and the cavities of normal size; (2) eccentric hypertrophy, which is also termed hypertrophy with dilatation or dilatation with thickening of the walls; and (3) simple dilatation or dilatation with thinning of the walls. A form was described by the older writers under the name of concentric hypertrophy, in which the cavities are diminished and the wall thickened, or, in other words, in which hypertrophy has occurred at expense of the cavities. Cruveilhier, however, pointed out, and observers are now generally of his opinion, that the reduction in the size of the cavities in such cases results from post-mortem rigidity and contraction. If the heart be macerated for some hours in warm water, it will be found that the cavity is of normal or even slightly excessive size. The form of enlargement, sometimes separately described, in which the cavity is dilated and the walls of normal thickness, is in reality a form of eccentric hypertrophy since the walls must be hypertrophied to maintain their normal thickness in spite of dilatation.

The degree of enlargement is always to be determined by the weight of the heart and the thickness of the walls. In the adult male the weight of the heart is from nine to ten ounces; in the female, from eight to nine. The left ventricle is from one-third to one-half inch, the right from one-sixth to a quarter of an inch; and the auricles about a line in thickness. In case of hypertrophy the weight of the heart is often from fifteen to twenty-five ounces, and sometimes much more. Stokes saw a case in which the weight was sixty-four ounces, Alonzo Clark one of fifty-seven, and Beverly Robinson one of fifty-three ounces. The thickness of the left ventricle has been known to reach an inch and a half, that of the right ventricle three-quarters of an inch, and the auricles may be double their normal thickness in cases of extreme hypertrophy.

Simple or pure hypertrophy is most frequently observed in the left ventricle, though it may occasionally occur in the right ventricle in cases of pulmonary disease. The auricles are never the seat of simple hypertrophy. On cutting into the heart the substance is found to be firm, of dark red color, and thicker than normal. The muscular trabeculæ within the cavity are often particularly hypertrophied, especially in the right ventricle. The shape of the heart is not usually much altered, though the apex of the organ is broader than normal. The size is necessarily less affected than when dilatation coexists with hypertrophy.

Eccentric hypertrophy, or hypertrophy with dilatation, gives rise to the largest hearts encountered. Some of these are well named by the term "cor bovinum" or "enormitas cordis" of the older authors. This form of enlargement is more frequent in the right ventricle and in the auricles than is pure hypertrophy; but it reaches the highest grades in the left ventricle, and may affect all of the cavities in the same heart. The size of the heart is much increased, and the apex is rounded so that the normal conical shape of the organ is to a large extent destroyed. The transverse diameter is much increased when both ventricles are enlarged, and when the right ventricle alone is involved the enlargement of the organ toward the right is a striking feature. The cavities are found dilated and the trabeculæ and papillary muscles hypertrophied, though at the same time flattened when dilatation is considerable. In the auricles the musculi pectinati are often increased in thickness. The muscular substance is firm and dark red, but in the late stages secondary degenerations are apt to occur, and render the muscle soft and lighter in color.

Simple dilatation is most frequently observed in the auricles and in the right ventricle. The cavities are sometimes enormously distended, as in the case of Stokes, in which the right auricle contained a pound of blood. I have seen the right ventricle so thinned and dilated that it presented the appearance of a large, bluish-colored tumor when the thorax was opened. The muscular substance may be so attenuated and atrophied that the endocardium is almost in contact with the pericardium. In ordinary cases, however, the muscular substance is merely thinned and softened. The shape of the heart depends upon the cavity involved, but for the most part assumes a rounded outline, similar to that of eccentric hypertrophy. Within, the septum may be seen to be pressed into the unaffected cavity. The orifices, especially the auriculo-ventricular, are frequently so distended that the valvular leaflets are incapable of closing them, and in this manner regurgitation of blood is permitted at the mitral or tricuspid valve, and secondary enlargement of the auricles ensues. The endocardium is usually thickened, and may be decidedly opaque in places. When the tricuspid valve is incompetent and the right auricle is much dilated, the venæ cavæ may be found enormously distended, sometimes reaching a diameter of two inches.

Very often blood-clots are found within the cavities and extending into the arteries and veins. Their presence is readily explained by the dilatation of the cavities and the slowing of the circulation, and occasionally fragments are detached and carried into the circulation as emboli.

Observers have differed in regard to the nature of hypertrophy of the heart, whether it be due to simple increase in the size of the individual fibres or to increase in their number, but it seems probable that both simple and numerical hypertrophy are usually present. There is, likewise, increase of the interstitial or interfibrillar connective tissue, which may contribute considerably to the cardiac enlargement. When fibroid overgrowth is marked, dilatation usually predominates over hypertrophy. In cases of great dilatation the muscle-fibres show the characteristic changes of the various myocardial degenerations, excepting the rare cases in which no microscopical alteration is discoverable in the heart.

Secondary pathological lesions are prone to follow hypertrophy and dilatation. Hypertrophy, though usually the result of arterio-sclerosis, often contributes to the increase of this condition when present or to its occurrence when not present. The explanation of this is found in the fact that blood-pressure is elevated suddenly with each contraction of the powerful ventricle, and may be constantly high. When marked arterial degeneration is present the cardiac hypertrophy sometimes aids in the production of aneurisms, and may directly occasion the rupture of a vessel in the brain or elsewhere. Secondary sclerosis of the valves of the heart is apt to result from the increased strain put upon them, but valvular incompetence is more frequently relative in nature, and due to stretching of the orifices. When the cardiac power fails and dilatation is marked, venous congestion of the various internal organs occurs, and leads to the same pathological changes as in cases of valvular disease. The embolic manifestations are likewise the same, though less frequently observed.

Symptomatology.—The symptoms present in a case of cardiac enlargement depend upon the relative degree of hypertrophy and dilatation. The former is so distinctly a compensatory process that, when it alone is present, symptoms are few and trifling, but, when dilatation supervenes, the cardiac compensation fails and evidences of stasis of blood are quickly manifested.

Certain subjective cardiac sensations may call attention to the heart even when hypertrophy is unaccompanied by dilatation. Among these are a feeling of weight or constriction at the præcordia or of general fulness of the thorax. The patient is often uncomfortable except when lying on the right side, and may be constantly conscious of the cardiac impulse, or there may be paroxysmal palpitation. Pain is rarely present, but dyspnœa may result from the direct pressure exerted upon the lungs. Throbbing of the vessels of the neck and tinnitus aurium are annoying symptoms frequently present. Sometimes the patient complains of hearing the heart-sounds as if they were produced within the ear, and sleep may be disturbed by this condition. All of these symptoms are aggravated by depression of the general health, and especially by neurasthenia or the over-use of tea, coffee, and tobacco. In such cases there may be decided disturbance of the patient's rest and comfort. The symptoms named are often ascribed to excessive hypertrophy, or, as we might say, to over-compensation, but in the majority of cases they are due to the general condition of the patient, and may be controlled by relieving the nervous excitability. Sometimes, however, it is probable that hypertrophy is excessive, and this is especially the case when no constant circulatory impediment is present, as in exophthalmic goitre or other conditions in which repeated nervous palpitation leads to hypertrophy. In these cases subjective sensations are apt to be decided.

When cardiac weakness and dilatation supervene, symptoms of failing circulation become established and may be exactly the same as those observed in the later stages of valvular disease. Indeed, the conditions within the heart may be the same as those which obtain in valvular disease, since the dilatation of the valvular orifices renders the segments incompetent and regurgitation of the blood ensues. The venous system becomes over-full and the various organs become congested. Dyspnœa, cough, and paroxysms of palpitation are common symptoms. The subjective sensations at the heart are often decided, and there may be pain radiating to the shoulder and left arm. More commonly palpitation is accompanied by vague apprehension without pain. The skin may be cyanosed, the veins distended, and when dilatation of the right ventricle is marked, undulation of the cervical veins is present, as in cases of tricuspid regurgitation from other causes. Headache or syncopal attacks give evidence of the disturbance of the cerebral circulation, and the patient's sleep is disturbed by unpleasant dreams. Delirium, Cheyne-Stokes respiration, and coma may occur, and occasionally a prolonged maniacal condition is observed. Enlargement of the liver, digestive disturbances, diarrhœa, and decrease in the secretion of urine result from congestion of the liver, the gastro-intestinal tract, and kidneys. Finally, œdema of the subcutaneous tissue and serous effusions in the internal cavities occur, as in valvular disease, and further add to the existing interference with the circulation.

The symptoms indicative of cardiac dilatation usually come on gradually, but may be extremely sudden in the course of infectious fevers or after severe muscular exertion or strain. In these cases the patient is at once seized with urgent dyspnœa and palpitation, and increasing cyanosis presages an early termination in a fatal issue.

Physical Signs.—The physical signs of enlargement of the heart depend upon the part of the organ involved and the relative degree of hypertrophy or dilatation present.

1. It will be best to consider first the signs indicative of enlargement of the left ventricle.

Inspection.—There is often a manifest bulging of the præcordial region, especially in children, and the intercostal spaces may be wider than normal. In older persons the left chest may seem fuller than the right, though no localized bulging is present. The cardiac impulse is seen below and to the left of its normal situation. Very often it occupies the sixth, seventh, or eighth interspace as far out as the anterior axillary line, or even beyond it. It is usually more diffuse than normal, and sometimes the whole præcordial region pulsates with each ventricular contraction. When dilatation is marked there is frequently a wavy pulsation occupying the third to the sixth interspace, and in extreme instances the impulse may be wholly invisible. Systolic retraction is occasionally noted along the left border of the sternum in cases of marked hypertrophy.

Palpation.—When hypertrophy is the predominating condition the cardiac impulse is of a peculiarly deliberate and heaving nature, and is decidedly forcible. Sometimes, as Hope first pointed out, the impulse is a double one, both beats being systolic, or more frequently one systolic and the other diastolic. The diastolic shock following the systolic contraction is probably due to sharp closure of the aortic valves, and may be distinctly palpable at the apex or over the root of the aorta. In cases of eccentric hypertrophy the apex-beat is more sudden or quick than in simple hypertrophy, and is apt also to be accompanied by diffuse pulsation over the ventricle. When dilatation is prominent the impulse becomes more and more weak, and may disappear entirely. More commonly a diffuse and wave-like or irregular pulsation is felt over the left ventricle, or it may be faintly visible without being palpable.

The pulse in pure hypertrophy or in hypertrophy with moderate dilatation is regular, full, and tense. Its character may be considerably modified if arterio-sclerosis obstructs the flow of blood, but in the absence of this condition it is usually sudden and throbbing in nature. When dilatation supervenes the pulse becomes weak and generally irregular, and is more frequent than in pure hypertrophy. The frequency, however, is dependent upon the general condition of the patient. During paroxysms of dyspnœa the pulse-rate may reach 160 or even 200; ordinarily it is not much above the normal. The sphygmographic tracing shows the suddenness and the fulness of the pulse in hypertrophy and the irregularity in dilatation, but cannot be regarded as distinctive in any case.

Percussion.—The most important fact obtained by percussion is the increased extent of cardiac dulness toward the left and downward. When dilatation coexists with hypertrophy the apex is more rounded than in health, and the transverse diameter of the dulness may reach extreme proportions.

Auscultation.—In pure hypertrophy the first sound of the heart is dull or heavy and prolonged, and may sometimes be reduplicated. Occasionally in young persons and in those in whom the ribs are elastic, the first sound is accompanied by a peculiar clinking sound, which was designated as the *cliquetis métallique* by Laennec. The second sound is accented, and in the aortic region is frequently ringing or reduplicated. The heart-sounds are often so loud that they may be heard considerably beyond the limits of the heart.

In proportion as dilatation advances and becomes predominant, the first sound becomes more and more like the second, until the two are quite indistinguishable in their quality. At the same time the normal pause following the second sound is shortened, and the character of the fœtal heart-sounds is therefore closely simulated. French writers have given to this condition the appropriate name of embryocardia. It is not invariably an indication of dilatation of the heart, but is always significant of extreme cardiac weakness. More frequently the peculiar canter rhythm, or bruit de galop, may be heard on auscultation. A systolic murmur is sometimes heard at the apex in cases of extreme dilatation, and is indicative of relative incompetence of the mitral leaflets. In such cases the similarity to valvular disease with secondary hypertrophy and dilatation is very close.

2. Enlargement of the right ventricle is rarely due to pure hypertrophy, and there is more frequently simple dilatation than in the case of the left ventricle.

Inspection may show a bulging or fulness of the lower part of the thorax, under the sternum, or there may be a distinct swelling in the epigastric region. The apex-beat may be displaced to the left, and may be seen outside the mammary line. When, however, the enlargement of the right ventricle is very great, the left heart is displaced from the chest-wall, and its impulse is therefore invisible. The impulse of the right ventricle is diffuse, and occupies the third to the sixth interspaces near the sternum. Not rarely pulsation may be seen to the right of the sternum. Cyanosis, repletion, and undulation of the veins of the neck and pulsation of the liver may be present in extreme cases when the ventricle is excessively dilated and the tricuspid leaflets incompetent.

Palpation.—The cardiac impulse is more diffuse and weaker than in cases of enlargement of the left ventricle. Occasionally a diastolic shock is felt in the second left interspace or over the heart, and is due to the sudden closure of the pulmonic valves under the excessive pressure of the lesser circulation.

Percussion.—The cardiac dulness extends far to the right. Not rarely the right limit is an inch beyond the sternal margin, and in extreme cases it may reach the mammary line.

Auscultation.—The first sound of the heart is generally louder than normal, and when dilatation is decided it becomes remarkably clear and sharp. In the latter case also there may be a systolic murmur of tricuspid incompetency heard with maximum intensity over the lower part of the sternum. The second heart-sound is sharply accentuated, or even reduplicated, in the second left intercostal space, but grows softer when the tricuspid valve becomes incompetent and the intraventricular pressure thereby reduced.

The pulse may remain normal as long as the right ventricle is able to maintain the pulmonary circulation ; when this fails the pulse is soft and weak.

3. Enlargement of the auricles is rarely found unassociated with involvement of one or the other ventricle. The physical signs are generally indistinct. A presystolic pulsation is occasionally seen or felt to the left or right of the sternum, and the cardiac dulness on percussion is found to extend high up along the borders of the sternum.

Diagnosis.—The points which lead to a diagnosis of enlargement of the heart are the increased area of cardiac dulness, the heaving or diffuse impulse, and the character of the sounds.

Increased area of dulness may, however, be produced by a number of other conditions, such as retraction of the lungs with greater exposure of the heart, consolidations of the anterior margins of the left lung, localized pleural effusions, pericardial effusion, and aneurisms, tumors, or other conditions which displace the heart forward against the chest-wall. Retraction of the lungs may result from pleural adhesions, phthisis, or cirrhosis of the lungs, and a similar exposure of the heart is found in flat-chested persons whose lungs are ill-developed and small. The diagnosis in such cases depends upon the careful study of the symptoms, of the heart-sounds, and of the effect of respiratory efforts on the area of cardiac dulness. The increased præcordial dulness resulting from consolidation in the anterior margins of the lungs and from sacculated pleural effusion is distinguished by the irregularity of the outline, by the associated auscultatory signs, and by the history of the case. Pericardial effusion may simulate extreme cardiac dilatation or the reverse, and the differentiation is practically very difficult in many cases. It has, however, been fully considered under Pericarditis.

Neurasthenic and hysterical patients frequently suffer extreme palpitation of the heart which may be mistaken for the palpitation of hypertrophy. The dulness, the character of the impulse, which is never heaving, and the sounds of the heart will furnish the basis for a differential diagnosis. It is to be remembered that repeated nervous palpitation leads to some hypertrophy.

Extreme dilatation of the heart with relative insufficiency of the valves occasions symptoms and physical signs practically indistinguishable from those of valvular disease. A relative murmur at the mitral or tricuspid orifice can only be distinguished from an organic one by the gradual disappearance under treatment as the ventricle regains its normal condition.

The determination of the relative degree of hypertrophy and dilatation present in a given case depends upon the symptoms and physical signs. If evidences of failing heart-power are absent, and the patient therefore presents few symptoms, hypertrophy is in excess of dilatation. The development of marked dilatation is attended by dyspnœa, rapidity and irregularity of the pulse, cyanosis, and other indications of failing circulation. The physical examination of the heart will show in pure hypertrophy the characteristic heaving impulse of rather limited area, without great alteration in the shape of the cardiac outline. On the other hand, when dilatation is marked the apex-beat becomes at first more sudden or "slapping," and later even imperceptible. There is, at the same time, a diffuse impulse over the entire præcordia, and the cardiac outline is more rounded, especially at the apex, than in health.

Prognosis.—Enlargement of the heart is a conservative process, with the purpose of maintaining the proper circulation despite obstructions. The prognosis therefore is mainly concerned with the question, how long the hypertrophy may be maintained without the supervention of excessive dilatation. The general health of the patient will therefore influence the prognosis. In a robust, healthy person hypertrophy may continue unimpaired for years, but in an anæmic, debilitated individual, and in one in whom the coronary arteries, together with the general arterial system, are the seat of sclerotic changes, the danger of myocardial degeneration and dilatation of the heart is ever present. Intercurrent febrile affections assume unusual importance in persons having hypertrophy of the heart, from their tendency to induce acute myocarditis and parenchymatous degeneration of the muscle.

When cardiac hypertrophy is once established, it usually remains persistent. It must, however, be admitted that in cases where the strain upon the heart is a temporary one, as in pregnancy, acute Bright's disease, hypertrophy from nervous palpitation, and the like, the muscle may resume its normal proportions when the cause of the hypertrophy ceases to be operative. It is especially in the young that the most marked instances are seen of recovery from hypertrophy, apparently due in part to the fact that the rapidly growing system overtakes and adjusts itself to the enlarged heart.

The prognostic significance of dilatation is exactly the reverse of hypertrophy, and in proportion as dilatation advances the prognosis gains in gravity. Acute dilatation may lead to a rapidly fatal issue. Even dilatation, however, may be recovered from. This is often the case in infectious fevers, in which moderate myocardial degeneration and dilatation lead to temporary weakness of the heart, from which the patient recovers under careful treatment and absolute repose. On the other hand, sudden death may occur in such cases if undue exertion be permitted.

Treatment.—The treatment of hypertrophy and dilatation is largely the same as that of valvular disease. In both the first dependence is to be placed on solicitous care in hygiene and the adjustment of every detail of the patient's life. In both the principles of treatment are—first, to aid in the establishment of such muscular power as may be necessary to overcome the impediment to the circulation and to institute measures which will maintain the compensation when established; and, second, to stimulate the heart and reduce peripheral resistance to the circulation when compensation begins to fail.

The patient should be directed to live quietly, to avoid physical and mental excitement or over-exertion. At the same time, it is to be remembered that judicious graduated exercise is essential for the maintenance of muscular tone. The scientific use of mild gymnastics or of out-door exercise in proper altitudes is always to be considered.

The diet is to be regulated so as to escape unnecessary taxation of the cardiac power. The food should be simple and small in quantity at each meal; tea and coffee must be taken sparingly. Alcohol and tobacco must be interdicted.

Subjective sensations, such as weight or constriction at the heart and palpitation, are controlled by nerve-sedatives like bromide of potassium or valerianate of ammonium. Excessive hypertrophy or over-compensation is more frequently present than in valvular disease, and may require the occasional use of tincture of aconite or veratrum viride. The bowels may be regulated, if necessary, by mild saline laxatives.

As soon as indications of failing heart-power become manifest the patient should be placed at complete rest and cardiac tonics or stimulants must be administered. Digitalis and strychnine are the most useful remedies under these circumstances. Digitalis must, however, be used with caution when sudden dilatation of the heart results from extensive myocardial disease. In these cases digitalis in moderate doses with strychnine is preferable to large doses of digitalis alone. It often happens that full doses of strychnine given hypodermically produce excellent results. Alcohol, ammonia, and other cardiac stimulants may occasionally prove useful, and tonics are serviceable in maintaining or restoring the muscular nutrition. In cases of sudden distention of the right heart and the venous system, venesection may be employed in the same manner and with the same precautions as in valvular disease.

ACUTE MYOCARDITIS.

SYNONYMS.—Carditis; Cardio-malacia; Abscess of the heart.

Definition.—Acute myocarditis is an inflammatory affection of the interstitial tissue of the heart-muscle, often accompanied by degenerations of the muscular fibres and by inflammation of the pericardium or endocardium. Virchow described under the head of "parenchymatous myocarditis" a condition in which the muscular fibres are filled with albuminoid granules, but this process is now recognized as one of the cardiac degenerations, and will be considered under that heading. This parenchymatous degeneration or cloudy swelling is frequently associated with myocarditis, but may occur alone, and is not in itself an inflammatory process.

Etiology and Morbid Anatomy.—Two forms of myocarditis may be distinguished—the diffuse and the circumscribed. The former of these is hyperplastic in nature; the latter is suppurative, giving rise to abscesses of the myocardium.

1. Diffuse myocarditis occurs most frequently in the course of the infectious fevers, especially in typhoid fever, diphtheria, scarlet fever, and small-pox. It is not uncommonly met with in articular rheumatism, with or without pericarditis or endocarditis, and in septic processes of all kinds. Councilman has recently called attention to gonorrhœa as an occasional cause. In some cases traumatism has been considered the cause, and occasionally apparently primary or idiopathic cases have been observed, but in these some minor infection like tonsillitis may have been overlooked.

In pericarditis and endocarditis the inflammation generally extends also to the muscular substance of the heart to the depth of a quarter or half a line, and in severe cases may involve a third of the thickness of the wall.

From our present evidence it would seem that myocarditis is generally infectious in nature, and dependent upon bacterial products rather than upon the micro-organisms themselves. The degree of fever does not necessarily determine the severity of the myocardial inflammation, for severe myocarditis may occur in cases in which the temperature is but slightly elevated.

Formerly myocarditis was considered a rare disease, and this is doubtless true of the severer grades, but recent investigations have shown that slight myocarditis is, on the contrary, exceedingly common in the infectious fevers named. According to Romberg, it may be found to exist in all fatal cases of diphtheria, in nearly all of scarlet fever, and in the majority of typhoid fever. Microscopical examination of the heart is, however, necessary to discover the morbid change in many of these cases.

The muscle of the heart is soft, and sometimes extremely so; its color is at first dark red and punctated with hæmorrhagic or deeply-injected areas; in the later stages it assumes a yellowish-red and mottled appearance, and may finally become light-yellow or grayish and friable. The cavities of the heart are frequently dilated, and in rare cases in which there has been localized myocarditis partial aneurism may result. In marked cases these appearances are decided, but in many the existence of myocarditis is discoverable only on microscopical examination. The minute changes consist in infiltration of the interstitial fibrous structure with round cells and free nuclei, and proliferation of the existing cells. The blood-vessels are engorged, and sometimes show inflammatory changes in their walls. The subpericardial and subendocardial tissue presents similar cellular infiltration. The muscular fibres themselves are usually involved in various degenerative changes. Most frequently they are filled with the minute albuminous granules characteristic of parenchymatous degeneration, the areas of more decided involvement showing distinct fatty degeneration. Occasionally hyaline transformation or atrophy with pigmentation may be observed. A peculiar form of transverse division or fragmentation of the fibres was described by Zenker, and later more fully by Renault and Landouzy. This *myocardite segmentaire* has been noted especially in cases of sudden death, but, as Zenker and Recklinghausen have recently maintained, is probably developed post-mortem. Its existence, however, may indicate some previous pathological condition of the muscle not demonstrable by our methods of research.

Diffuse myocarditis affects the left ventricle more frequently than the right, excepting when fœtal in origin. In the latter case the right ventricle alone may be involved. The process may be uniformly diffused or may affect scattered areas in different regions. Sometimes it is confined to the muscular ring surrounding the mitral or tricuspid orifices, or to the papillary muscles, and thus induces derangements of the valvular mechanism. It may terminate in complete recovery by resolution, in chronic fibroid myocarditis, or very rarely in suppuration. Endocarditis and pericarditis may be secondary to myocarditis, but are more frequently antecedent to it.

2. Localized or suppurative myocarditis results from infection with the micro-organisms of suppuration, which are not rarely demonstrable in the lesions. These organisms may reach the heart-muscle as emboli in the branches of the coronary arteries, coming from a primary septic focus in puerperal septicæmia, osteomyelitis, suppurative phlebitis, malignant endocarditis, and the like, or they may invade the heart directly from malignant endocarditis or pyopericardium. Suppurative myocarditis may, however, also arise in the course of acute rheumatism, typhoid fever, scarlet fever, or diphtheria, though much less frequently than does the diffuse, hyperplastic form. In these cases the primary focus of suppuration may be difficult to determine.

There are seen on section of the heart-wall numerous small yellowish-white spots or streaks, varying in size from a pin's head to a grain of wheat, which on microscopical examination are found to consist of a central mass of micrococci surrounded by leucocytes. Later these areas grow larger, even to the size of a small cherry; they become surrounded by a hæmorrhagic zone; and finally assume the character of small abscesses scattered through the substance of the heart. In one case Roth removed nearly an ounce of pus from the abscess-cavities. Microscopically, the muscle-fibres adjoining these larger foci are seen to have become degenerated, and they may be completely destroyed.

The most frequent seats of abscesses of the heart are the anterior wall of the left ventricle near the apex and the septum, but they have been observed in the auricles and in the right ventricle. The abscess, if situated in the ventricular wall, may rupture into the pericardial sac and produce suppurative pericarditis, or into the cavity of the heart and lead to malignant endocarditis and general septicæmia. Purulent pericarditis and malignant endocarditis may, however, occur without visible rupture. Aneurismal dilatation may occur at the point weakened by the abscess-cavity, and rupture of the heart may result. Localized softening of the heart-muscle may occur in an area of anæmic necrosis from embolism without suppuration, and closely simulates the genuine abscess. It must be remembered that the tendency to subsequent cicatrization, however, distinguishes such lesions. An abscess in the septum ventriculorum may lead to an aneurismal dilatation, the convexity being in the right ventricle, or the abscess may rupture into either ventricle. Occasionally complete perforation of the septum has been observed, and in a few sinuous communications have been found extending from the left ventricle into the right auricle.

Suppurative myocarditis usually terminates in a fatal issue as a result of the cardiac condition or of the primary disease. In some cases, however, the abscesses are encapsulated, and the pus becomes inspissated and cheesy, or calcareous nodules may remain as the only evidences of their previous existence.

Symptomatology.—The symptoms of either form are indefinite and are generally obscured by the primary disease. There may be vague subjective sensations, such as constriction or pressure, referred to the cardiac region, and occasionally even decided pain. The most important symptoms, however, are those indicative of cardiac weakness. Arrhythmia is a frequent and significant symptom when occurring in the course of rheumatism or the infectious fevers. The pulse is generally accelerated, and there may be paroxysms of great rapidity, the pulse-rate sometimes reaching 200 in the minute. Dyspnœa and cough, with anxiety and general depression, may occasionally be prominent indications.

Sudden dilatation of the cavities may supervene, and occasion extreme disturbance of the circulation. Endocarditis and pericarditis, whether preceding or following myocarditis, mask its symptoms, but in such cases the disturbance of the cardiac power is greater than would occur in uncomplicated endocarditis or pericarditis.

Suppurative myocarditis may give rise to various embolic manifestations when the abscess ruptures into the heart. In such cases purpuric and pustular skin eruptions and splenic enlargement have occasionally been observed.

The fever present in the primary disease usually rises somewhat with the development of myocarditis, and may be septic in type in the case of abscess.

Physical Signs.—On auscultation the rapidity and irregularity of the action of the heart will be noted, and the first sound is remarkably weak. Murmurs are frequently developed as a result of dilatation and relative insufficiency of the valvular segments or of relaxation of the muscle at the orifice. The most frequent is a systolic bruit indicative of mitral regurgitation. Dilatation of the heart may occasion increase of the cardiac dulness.

Diagnosis.—The diagnosis can never be made with certainty, but signs of great weakness of the heart, with a weak first sound or a systolic murmur, would point to the existence of the cardiac complication. The suppurative form may be suspected when the heart grows suddenly weak in the course of pyæmic diseases, and if embolic manifestations supervene the diagnosis would be more certain. Autopsy, however, brings many disappointments to those who hazard positive diagnoses in these cases.

Prognosis.—Slight diffuse myocarditis is completely recovered from in most cases, but the severer grades may lead to a fatal termination in a few days or a week. Recovery may ensue even in the suppurative form, as the calcareous remnants in the heart-walls would indicate. The sudden death occurring in infectious fevers, particularly in diphtheria, is often the result of myocarditis.

Treatment.—Absolute rest must always be insisted upon. The patient must not rise from the reclining posture for any purpose. Quinine and strychnine are exceedingly valuable as general muscular tonics, but heart-stimulants, especially digitalis, should be used with care, and never in large doses. Clinical experience has demonstrated the value of alcohol in the myocardial diseases of infectious fevers, and this remedy should therefore be given as soon as the first sound grows weak. In case of cardiac abscess large doses of digitalis might favor a fatal rupture of the heart. When uneasiness is experienced in the præcordial region, cold applications will frequently bring prompt relief. Cloths saturated with ice-water, an ice-bag, or Leiter's coils may be used for this purpose.

In every case of infectious fever in which cardiac weakness is observed care must be taken to avoid undue excitement or exertion for a long time after subsidence of the fever.

CHRONIC MYOCARDITIS.

SYNONYMS.—Fibroid heart; Chronic interstitial myocarditis; Cardiosclérose.

Definition.—Chronic myocarditis is a chronic inflammatory and hyperplastic process affecting the interstitial connective tissue of the heart and leading to induration of its substance. Like acute myocarditis, it is usually accompanied by certain degenerative changes in the fibres themselves.

Etiology.—The most frequent etiological factor is sclerosis of the coronary arteries, and the association of renal disease, arterio-sclerosis, and fibroid myocarditis is therefore commonly met with in persons of advanced age who have indulged freely in alcohol and a generous table or who have contracted syphilis. The influence of excessive bodily exertion may also be apparent in certain cases. Fibroid disease of the heart probably develops in many of these cases primarily—that is, in consequence of the same tendency to fibrous overgrowth which is manifest in the arteries and the kidneys—but in most cases the cardiac condition is secondary to the coronary sclerosis. The immediate cause of the myocardial affection in these latter is the stoppage of the circulation by thrombosis, to which the narrowing of the vessels predisposes. This results in the development of infarctions which are gradually transformed into sclerotic areas. In other cases the insufficiency of the blood-supply through the occluded vessels may, without thrombosis or infarction, induce a low grade of morbid action with gradual tissue-changes.

Fibroid myocarditis may, on the other hand, be present without primary affection of the coronary arteries, though less commonly. This occurs in a variety of infectious fevers which induce, first, diffuse acute myocarditis, and, secondarily, fibroid overgrowth. Acute articular rheumatism is prominent among the diseases acting in this manner. The resulting cardiac condition is rarely marked in such cases. Certain poisons may likewise act upon the myocardium and induce slow sclerotic changes. Among these are alcohol, the syphilitic poison, and probably toxic agents resulting from improper metabolism, as in gout.

Myocarditis is frequently found associated with pericarditis and endocarditis, and may be present in the underlying muscle to a considerable depth, especially in cases of adherent pericardium.

In many cases of chronic valvular disease microscopic study of the heart-wall discloses slight or decided grades of chronic myocarditis, the presence of which is accounted for by the high degree of coronary sclerosis present in some instances, or simply by the interference with proper nutritive supply occasioned by the valvular affection in others. A limited amount of fibroid change is frequently found in cases of mitral or tricuspid valvulitis affecting the tips of the papillary muscles. In these cases the myocardial involvement results directly from extension of the inflammation along the chordæ tendineæ to their muscular attachment.

Age and sex exercise no decided predisposing influence, but coronary disease and therefore fibroid myocarditis, more commonly affects men beyond forty years than women or young persons of either sex. It is probable that old age does in itself play a part in the production of the disease, since sclerotic changes are elsewhere observed with advancing years; but the tendency to marked sclerosis of the arteries in the aged is the important etiological factor. Chronic myocarditis sometimes arises in fœtal life, and may occasion congenital malformations, particularly stenosis of the conus arteriosus of the right ventricle.

Morbid Anatomy.—The anatomical changes may be diffuse or may be limited to certain parts of the muscular substance. The portions most frequently affected are the wall of the left ventricle, the papillary muscles, and the septum, and when the process is diffuse these parts are more extensively involved than others. A localized area of chronic myocarditis is not infrequently detected in the anterior wall of the left ventricle near the apex, and occasionally a zone of fibroid induration and contraction leads to stenosis of the sinus of the pulmonary, or more rarely the aortic, orifice.

The heart in chronic myocarditis is enlarged, and its weight may be considerably increased. In a case before mentioned Quain found the weight of the heart to be forty ounces. The increased size results from hypertrophy and dilatation of the walls in addition to the fibroid overgrowth. The degree of hypertrophy and dilatation depends upon associated conditions, particularly upon the state of the general arterial system. When marked arterio-sclerosis is present, hypertrophy of the heart-muscle may be decided. Sooner or later, however, dilatation of the cavities occurs, and occasions serious secondary disturbances of the circulation. In adherent pericardium dilatation is sometimes extreme.

The heart-wall is firm and cuts with distinct resistance to the knife. Certain areas may be almost tendinous in appearance, and generally opaque, white, or grayish spots or streaks are discoverable in the muscular substance. The endocardium may be thickened, and grayish-white spots or streaks are generally visible through it or the pericardium. Larger areas of induration may be seen near the apex or elsewhere. The papillary muscles are frequently involved, and may be decidedly indurated and contracted.

The minute changes consist in overgrowth of the interfibrillar connective tissue, with development of fibrous tissue. These changes may be uniformly distributed through the substance of the heart when some intoxication, as by alcohol, or some general disturbance of the cardiac nutrition, has led to the myocardial disease; or they may be seen in circumscribed areas when embolic or thrombotic occlusion of branches of the coronary arteries has occasioned anæmic infarction and subsequent sclerosis. In either case the microscope reveals masses of wavy fibrous tissue between the muscular bundles, and often slow degeneration or atrophy of the fibres themselves. Various grades of parenchymatous or fatty degeneration and vacuolation may be present, and when the pressure exerted by the new-formed fibrous tissue is very great, atrophy of the fibres may be extreme. The terminal branches of the coronary arteries are generally more or less sclerotic and narrowed, and in syphilitic cases particularly obliterating endarteritis is common. The ends of the papillary muscles and the chordæ tendineæ are frequently converted into tendinous cords.

Associated morbid changes in other organs are more frequently due to the arterio-sclerosis than to the cardiac disease; but when the power of the heart grows insufficient, the same congestions and degenerations are met with in the internal organs as are seen in valvular disease.

Aneurism of the heart is commonly due to localized cardio-sclerosis. The inelastic fibrous tissue gradually gives way before the intracardial pressure, and saccular dilatation results.

Symptoms.—In many cases no symptoms are present, and the fibroid disease of the heart is discovered post-mortem. The latency of these cases is due to the compensatory hypertrophy of the heart and to the absence of disturbance of the cardiac innervation.

Generally, however, failure of the heart-power to maintain the circulation is sooner or later manifested, though the symptoms vary so widely in different cases as to deprive the disease of any definite characters. Dyspnœa on slight exertion is frequently an early symptom, and may be accompanied by palpitation and other subjective indications. Constriction or pressure at the heart is very commonly experienced, and not rarely there is decided pain of anginal character. Throughout the entire disease paroxysms of angina pectoris may be the only symptoms calling attention to the heart. In the intervals the patient may seem completely well, but disturbances of the action of the heart are generally persistent after the anginal paroxysm has subsided. Cardiac arrhythmia is very common, and may assume any of its various forms. Perhaps the most common is slight intermission with inequality of the successive beats of the pulse. Cases are, however, at times observed in which the rhythm is constantly regular. The pulse-rate is generally decreased, and is frequently reduced to forty or fifty beats per minute. An extreme case is recorded in which the rate was said to have been but eight in the minute. In some instances, in which slowing of the pulse is constant and marked, patients complain of great palpitation during anginal attacks even when the heart-beats are far below the normal frequency.

Sudden failure of the cerebral circulation is denoted by attacks of syncope, which come on without warning or more frequently when the patient has been exhausted by undue exertion. More rarely persons suffering with fibroid heart are prostrated by pseudo-apoplectic attacks, coming on after some mental or physical excitement, and sometimes leading to sudden death. Such attacks may occur in persons who have experienced no previous symptom of cardiac disease, and are easily mistaken for manifestations of cerebral hæmorrhage. When the cavities of the heart begin to dilate, the evidences of failing circulation become more pronounced. Very often paroxysmal dyspnœa or cardiac asthma is observed. Later congestive enlargement of the liver, decreased excretion of urine, and derangements of the gastro-intestinal tract denote the progressive failure of the cardiac power. Throughout the entire course of the disease the patient may present great weakness and cloudiness of the mind, and in the latter periods may become either excessively somnolent or wakeful. Delirium and chronic mania are occasionally observed.

Physical Signs.—In some cases enlargement of the left ventricle is detected by the outward and downward displacement of the apex-beat and by increase of the cardiac dulness in the same directions. The heart-sounds at first may be clear and loud, but soon become decidedly weak and muffled. The tension in the lesser circulation is often elevated, and in these cases the second sound is accentuated, or even reduplicated, in the pulmonary region. A systolic murmur of mitral insufficiency may result from contraction of the papillary muscles and chordæ tendineæ or from dilatation of the ventricle and the valvular orifice. It is heard at the apex and toward the left axilla, but rarely has the wide transmission of the murmurs of organic mitral regurgitation.

The action of the heart may be extremely irregular, and the sounds of varying loudness. The irregular rhythm called bruit de galop is occasionally heard, but is more properly attributable to the secondary dilatation than to the indurative myocarditis.

Diagnosis.—In some cases the latency of the symptoms may prevent suspicion of the disease, though the cardiac changes are marked; in others the evidences of cardiac weakness are clear enough, but the differential diagnosis from valvular disease, from hypertrophy and dilatation, and from fatty degeneration is far less certain.

As a rule, the absence of murmurs will distinguish valvular disease, but it is to be remembered that in the last stages of valvular affections the murmurs may disappear completely, and, on the other hand, the accidental or relative valvular murmurs in fibroid myocarditis may be indistinguishable from those of organic disease. In one case under my observation there was a loud aortic systolic murmur dependent on roughening of the lining of the aorta, but no constriction of the orifice. The pulse was small and infrequent, and the symptoms of failing circulation were pronounced. In such rare cases the diagnosis is attended with great difficulty.

The distinction of fatty degeneration or hypertrophy and dilatation from fibroid heart may be quite impossible. Anginal attacks are more common in chronic myocarditis than either of the other conditions; but it is impossible to distinguish in some cases, and the three conditions are often found associated. The pseudo-apoplectic attacks of chronic myocarditis resemble seizures of true apoplexy in some cases, and can only be distinguished by the history and by the progress of the case.

Prognosis.—The prognosis of fibroid heart is entirely unfavorable *quoad valetudinem,* but favorable *quoad vitam.* The patient may live for many years with considerable coronary sclerosis and fibroid overgrowth of the heart. The length of life and the degree of comfort of the patient will depend upon the surrounding circumstances. If arterial or renal sclerosis be present or the labor of the patient severe, the heart-power is apt to fail more quickly than when the circulation is little impeded and the heart is severely taxed. In some cases death occurs suddenly after an anginal attack, from syncope, or in one of the pseudo-apoplectiform attacks before mentioned.

Treatment.—The treatment of fibroid heart is practically the same as that of hypertrophy and dilatation. When once established, it is improbable that any remedy has power to remove the fibrous tissue. In syphilitic cases, or in others also, iodide of potassium may be used, but probably has little value. The patient's general nutrition should be carefully studied, and tonics like arsenic and iron be administered in case of anæmia.

The subjective symptoms referred to the heart are frequently urgent, and require palliation. An ice-bag or warm applications over the heart will frequently give relief in cases of cardiac oppression or constriction. When the sense of palpitation is present, nerve-sedatives may be useful, or, if the heart be really excited in action, a few drops of tincture of aconite may be employed. In the cases of purely nervous palpitation bromide of potassium, valerianate of ammonium, and asafœtida are remedies of which we may make frequent use. More severe subjective sensations, and especially anginal pain, will require more active measures. Sometimes Hoffmann's anodyne or hot milk containing whiskey or brandy may bring relief; in others nitrite of amyl or morphine are required, as in true angina. The morphine is best given hypodermically and in combination with atropine. When blood-pressure is high and anginal attacks prone to occur, the continuous administration of nitro-glycerin in small doses is generally beneficial, and may prevent the painful paroxysms.

Signs of cardiac weakness demand the use of some form of cardiac stimulant. In cases in which the heart's action is rapid, irregular, and weak digitalis is of great value, but when the pulse is infrequent this remedy must be used with care. Marked arterio-sclerosis always requires that digitalis be used sparingly, lest undue increase of the blood-pressure occasion increased distress or favor rupture of a vessel in the brain. When digitalis seems undesirable, nux vomica or strychnine, nitro-glycerin, ammonia, and alcohol are useful cardiac stimulants. Strychnine is especially valuable on account of its tonic influence on the muscles, and in combination with quinine and extract of opium acts well in cases in which the general nutrition is poor and painful anginal attacks are frequent.

The syncopal attacks of fibroid heart must be treated in the same way as syncope from other causes. The clothing of the patient must be loosened about the neck, the head should be lowered, and stimulants administered. The patient may be revived sufficiently by the odor of ammonia to take some hot whiskey or brandy. In bad cases the hypodermic injection of ether, ammonia, or brandy may be necessary to stimulate the cardiac action.

The general life and habits of a person suffering from chronic myocarditis should be the same as in cases of valvular disease, but even greater care will be necessary to guard against over-exertion. The diet should be plain and simple. Tea and coffee must be used sparingly, and tobacco not at all. The advisability of the habitual use of some alcoholic drink at meal-times is, to a large extent, dependent upon the previous habits and the age of the patient. Elderly patients often take a little wine with advantage, but malted drinks are nearly always harmful. As a rule, it is best to save alcohol, like other cardiac stimulants, for times of special necessity.

DEGENERATIONS OF THE MYOCARDIUM.

Under this heading will be described those alterations of the heart-muscle which result from nutritive disturbances and impair the functional power of the organ. Among these degenerations are recognized anæmic necrosis, calcification, amyloid, hyaline, and parenchymatous degeneration, and fatty changes, infiltration and degeneration. The first four are generally unimportant conditions, and are uncommon, so that they may be dismissed with but a brief description. Parenchymatous degeneration and the fatty changes are, on the other hand, most important.

ANÆMIC NECROSIS.—This name is used to indicate the localized degenerative changes which take place in the myocardium as a result of embolic or thrombotic occlusion of branches of the coronary arteries. Most frequently sclerosis and atheroma of these vessels are the cause of thrombosis and subsequent anæmic necrosis, but the arteries may be entirely normal when the infarction results from embolism. Anæmic necrosis is generally met with in the distribution of the anterior coronary artery, and therefore in the left ventricle or septum ventriculorum. In the case of embolism, which is much less common than is thrombosis, the lesion is usually single, and is situated near the apex of the heart anteriorly. The area from which the blood is cut off becomes anæmic, gray, white, or yellowish in color, and is irregularly wedge-shaped or more frequently rounded in outline. Subsequently it may break down into a mass of softened and degenerated detritus, and on this account the process is sometimes called *myomalacia cordis.* The softened area may give way and rupture of the heart result. In other cases, however, softening does not occur, but instead the infarct becomes of hyaline appearance and gradually grows sclerotic. A heart may thus become extensively fibrotic by numerous small infarctions with subsequent sclerosis. The minute changes consist in various degenerations of the muscle-fibres. Very commonly they become more or less granular, losing their customary striated appearance, and finally break down into a mass of granular material. The degenerated fibres stain poorly, and the nuclei are obscured by the granulation. Neighboring fibres may be highly fatty. In other cases the muscle-cells become of hyaline appearance, and finally the area is transformed into sclerotic tissue.

The symptoms of occlusion of the coronary arteries sometimes come on with great suddenness and severity, and may terminate in speedy death. Sometimes anginoid pain, with failure of the cardiac power, is the decided, though indefinite, indication of the affection of the myocardium. There may be but one paroxysm, ending in death, or there may be repeated attacks of lesser severity, and doubtless the anginoid attacks in the course of chronic myocarditis are sometimes due to the sudden occlusion of a branch of the coronary artery. Asthmatic symptoms may be associated with cardiac pain or may be independent of such.

Calcification in the myocardium is a rare condition. Occasionally cretaceous nodules are found in the heart-muscle, whose presence is explained by an old purulent myocarditis, with subsequent inspissation of the pus and calcification. Calcareous infiltration of the muscle-fibres themselves has also been observed, but is extremely rare.

Amyloid Degeneration is sometimes found affecting the connective tissue of the myocardium, and results from the same causes which induce amyloid disease of other organs. Among these causes are prolonged suppurations, tuberculosis, syphilis, malaria, and cachexia due to other influences. The degeneration affects the connective tissue, and is rarely sufficiently marked to cause gross alterations in the appearance of the muscle. In a few cases, however, the condition has been recognized by macroscopic examination.

Hyaline Degeneration is sometimes associated with amyloid degeneration, resulting from the same causes. It is recognized by the glassy or waxy transformation of the connective tissue, and is usually limited to scattered areas. In other cases a hyaline condition of the fibres themselves is observed, notably in infectious fevers and in areas of anæmic necrosis. The fibres swell and become of homogeneous appearance, losing their striations and their power of receiving stains. The hyaline condition of the muscle-fibres in febrile diseases was first described by Zenker, and pathologically consists in a swelling of the cement-substance uniting the contractile elements.

The symptoms of this condition are those of parenchymatous degeneration, with which it is usually associated. Usually only scattered fibres, here and there, are affected, the majority presenting the appearance of cloudy swelling.

PARENCHYMATOUS DEGENERATION.—This condition, also called cloudy swelling, was formerly regarded as a form of inflammation, and termed parenchymatous myocarditis. It is met with in the course of the infectious fevers, particularly in diphtheria, typhoid fever, and scarlet fever. The degree of temperature itself is unimportant, since advanced degeneration may occur in cases in which the temperature has been little elevated. Cloudy swelling is also associated with pericarditis and endocarditis, affecting the adjacent myocardium.

The changes are most marked in the left ventricle, but may affect any part of the heart. The myocardium becomes pale in color, often of a peculiar turbid appearance, and is always softer than normal, so that the term "cardio-malacia" or "myomalacia" has also been applied to this condition. Sometimes the walls are extremely flabby and the cavities dilated.

Microscopically, the muscle-fibres are filled with numerous fine granules, whose albuminous nature is recognized by their solubility in acetic acid and dilute solutions of caustic potash. The striæ and nuclei are often obscured by the granular condition, but, when the specimen is treated with acetic acid, swelling and multiplication of the nuclei may be apparent. The interstitial connective tissue may be unaffected, or there may be some proliferation and infiltration with round cells between the muscle-fibres. In infectious fevers it is customary to find more or less interstitial myocarditis associated with cloudy swelling of the fibres, but either may exist alone. The parenchymatous changes occur earlier and reach a higher grade in diphtheria than in any other of the infectious fevers. Zenker's hyaline transformation may be found in places in a minority of the cases, but rarely becomes marked. Parenchymatous degeneration tends to further change into fatty degeneration, and intermediate stages are difficult of interpretation excepting by the chemical tests suggested. Fragmentation of the muscle-fibres—the *état segmentaire* of Renault—is also observed in certain cases. It may depend upon chemical or vital alterations of the fibres at present unrecognizable, but the fragmentation itself doubtless occurs in the death-agony or post-mortem.

The symptoms of parenchymatous degeneration are exactly the same as those of acute diffuse myocarditis. The heart's action becomes weak and rapid, and frequently irregular. The impulse is weak or imperceptible, and the sounds become more or less altered. The first sound is generally much weaker than normal, but often quite clear. The long pause may be shortened, and the fœtal rhythm or embryocardia is then detected. Imperfection of the muscular mechanism or acute dilatation may lead to a soft systolic murmur at the apex. Occasionally the pulse-rate is reduced to forty or fifty. The pulse is always weak and frequently dicrotic.

FATTY HEART is the name by which are designated two quite distinct pathological conditions—fatty infiltration and fatty degeneration—the former being of the nature of a deposit of fat upon and in the substance of the organ, the latter of a direct transformation of the protoplasm of the muscle-fibres into fat. Though thus distinct in their pathological nature, fatty degeneration and infiltration are frequently associated, and when occurring singly lead to similar clinical features, so that there is a manifest advantage in the general term "fatty heart."

Fatty Infiltration.—Cor Adiposum; Adipositas s. Obesitas cordis. In the normal heart there is a certain amount of adipose tissue in the visceral layer of the pericardium, particularly in the furrows and along the line of the blood-vessels. The yellow bands of fat bordering either side of the coronary vessels to their minutest subdivisions form a characteristic picture. In cases of general obesity this normal layer of fat is increased, and other portions of the pericardium and the heart become invaded at the same time. There may be a heavy deposit in the parietal pericardium and outside the sac in the anterior mediastinum; the layer directly covering the heart itself may become a half inch or inch in thickness; and the organ may thus be deeply imbedded in a mass of adipose tissue. On section of the heart-wall trabeculæ of fatty tissue may be seen passing in between the muscular fibres from the pericardium even to the endocardium, and very rarely the trabeculæ and papillary muscles themselves may be invaded. The deposited fat has usually a bright-yellow or whitish color, but occasionally has a peculiar icteric hue. Microscopically, the interstitial spaces between the muscle-fibres are densely packed with large oil-containing cells, of the characteristic appearance of adipose tissue. The heart-muscle itself is sometimes healthy; oftener, in cases of considerable deposit, the fibres undergo various atrophic and degenerative changes from pressure. Microscopically, the fibres may be full of albuminous granules or minute oil-drops, or their size may be reduced by simple or brown atrophy without degeneration. The myocardium in the latter cases may be reduced to a narrow rim of brownish color, and in places the pericardium and endocardium may be almost or quite in contact. The cavities may be considerably dilated, and rupture of the heart is a not uncommon result.

The causes of fatty infiltration of the heart are those which induce general polysarcia. Very frequently there is a manifest hereditary tendency, but over-eating and drinking and sedentary habits are powerful determining factors. In women who are sterile or have amenorrhœa, and after the menopause, there is sometimes a marked tendency to polysarcia. In general, fatty infiltration of the heart affects persons of advanced years, and is considerably more common in men than in women. Aside from the cases in which the cardiac condition attends or is part of general obesity, there is a form of fatty infiltration of the heart in which the patient is emaciated. In the general atrophy of old age or of cachexiæ there are sometimes considerable deposits of fat about the internal organs, and the same may be seen in cases in which a particular organ, as the heart, for example, is wasted and reduced in size.

The symptoms are indefinite. Corpulent patients often complain of difficulty in breathing in ascending elevations or walking with more than customary rapidity. This dyspnœa may result from cardiac weakness or it may be due to the mechanical effects on respiration of increased bulk. Cough may be present with or without bronchitis. The pulse is generally more rapid than normal, and decidedly weak. Sometimes, on the contrary, the pulse is infrequent. Anginoid attacks and less urgent subjective cardiac sensations are not infrequent.

The physical examination of the heart is attended with great difficulty on account of the thickness of the chest-walls. The cardiac dulness may be increased on account of the deposit or of dilatation of the cavities, but fatty accumulation in the anterior mediastinum also leads to apparent enlargement of the heart. The apex-beat is weak and often quite imperceptible; the heart-sounds are muffled and weak; and occasionally a systolic murmur may be detected at the apex.

Fatty infiltration may lead to sudden death by rupture of the heart.

Fatty Degeneration is a much more serious condition, in that the muscle-fibres are themselves transformed into fat, and the functional power of the organ therefore seriously impaired.

It may affect the heart altered by previous disease, or may occur primarily in the healthy myocardium. The occurrence of fatty degeneration in the hypertrophied heart of valvular disease or of general arterio-sclerosis has been referred to, and explained by the improper nutritive supply furnished the heart in these conditions. The degenerated myocardium is unable to maintain the circulation, or, as we say, compensation fails. Still further interference with cardiac nutrition results, and in turn greater cardiac degeneration ensues, until high grades of fatty degeneration are attained. The association of fatty degeneration with valvular disease, and its relation to failure of the compensation, have, however, been magnified. In many cases of decided failure of compensation no fatty degeneration is found when the heart is examined post-mortem. Sclerosis of the coronary arteries plays an important part in disturbing the cardiac nutrition, and leading to fatty degeneration as well as to fibroid myocarditis. In the infectious fevers, if prolonged, parenchymatous degeneration may give place, or rather advance, to fatty metamorphosis. This is especially frequent in diphtheria and typhoid fever, and may reach extreme grades, though usually only scattered areas or fibres are involved.

The previously healthy heart may become fatty as a result of many conditions which deteriorate the quality of the blood, or as a result of toxic agents. Probably the most extensive fatty degeneration seen in any condition occurs in cases of chlorosis or pernicious anæmia. Less decided degeneration is seen in phthisis, cancer, Addison's disease, and various other secondary anæmias. Sometimes, after great loss of blood by hæmorrhage, extreme and fatal fatty degeneration is rapidly developed. Among the poisonous agents which lead to fatty degeneration may be reckoned phosphorus, arsenic, and alcohol. In the case of the former two the condition may be suddenly developed, but in the latter only after long periods of intemperance is the cardiac degeneration established, and often only after primary coronary sclerosis.

The causes of fatty degeneration, like those of infiltration, are met with especially in advanced life and in the male sex.

The appearance of the heart varies with the degree of degeneration and with its extent. The first evidence is usually to be seen in the presence of yellowish striæ and points under the endocardium, especially in the papillary muscles and trabeculæ. The general myocardium may be healthy. In diffuse and extreme degeneration, such as occurs in phosphorus-poisoning and pernicious anæmia, the entire muscular substance may be of a light-yellowish appearance and very flabby and friable. Sometimes the substance tears with extreme ease, and it may seem remarkable that the walls were able to withstand the slightest intracardial pressure. Occasionally the color of the muscle is rather brownish from coexistence of brown atrophy, but this condition is usually limited to small areas. The cavities of the heart may be widely dilated, and the orifices so stretched that the valve-segments are incapable of closing them. Sclerosis of the coronary arteries and of the root of the aorta is frequently associated, and the former vessels may stand out prominently as tortuous cords. Fatty degeneration not infrequently leads to rupture of the heart, though the proportion set by Quain—24 in 83 cases—is far too high. Doubtless in some of his cases localized embolic or thrombotic softening was mistaken for fatty degeneration.

Microscopically, the fibres are filled with small granules and oil-drops, whose nature is apparent from their brown discoloration when osmic acid is applied and from their insolubility in acetic acid. Albuminous granules are frequently present in the early stages, and are recognized by the chemical tests mentioned under Parenchymatous Degeneration. The striæ and nuclei of the muscle-cells become indistinct, and finally the fibre is densely filled with small globules of oil. In certain of the fibres there may sometimes be seen, near the ends of the nuclei or evenly distributed throughout, the characteristic brownish granules of brown atrophy.

The symptoms of fatty degeneration of the myocardium are scarcely to be distinguished from those of chronic myocarditis in many cases. In other instances cardiac disease of any kind may be unsuspected until post-mortem examination reveals a high grade of fatty degeneration.

As a rule, there are unmistakable evidences of cardiac weakness. The pulse becomes weak, and often is irregular. The rate may be increased, but is not rarely greatly reduced in frequency, as in fibroid myocarditis. Attacks of palpitation and other subjective sensations, such as pressure or a feeling of constriction, may be experienced, and anginoid paroxysms are sometimes severe. The latter may be frequently repeated, and may alternate with attacks of extreme dyspnœa or cardiac asthma. These painful and asthmatic paroxysms may come on spontaneously during the night or day, or they may be induced by exertion or a full meal.

The patient is generally extremely weak and depressed, mentally as well as physically. There is often more or less emaciation, in contradistinction to the condition met with in fatty infiltration, so that Paget's designation of it as a "lean degeneration" is not inapt. The skin may be of peculiar pallor or whiteness, especially about the nose and under the eyes. The peculiar yellowish or opaque zone at the upper or lower margin of the cornea, known as the arcus senilis, is frequently present, but may be unassociated with fatty heart in elderly persons.

With increasing cardiac weakness the peripheral circulation fails, and various indications of failing heart-power are observed. Disturbance of the cerebral circulation is manifested by syncopal attacks or by pseudo-apoplectic seizures. These may come on wholly without warning, or may be foreshadowed by extreme slowness of the pulse, by nausea, or by vague foreboding. The apoplectiform attacks may be slight, consisting of but momentary loss of consciousness with fixation of the body and occasionally slight twitching, or the patient may pass into complete coma. On return of consciousness a temporary paresis of the muscles may be observed, but sometimes cerebral hæmorrhage may occur during the attack and leave the patient permanently hemiplegic. Cheyne-Stokes breathing may occur with or apart from the symptoms just named, and sometimes persists for many days or weeks. During its existence the patient is more or less stuporous or comatose. Cheyne-Stokes breathing, however, accompanies many conditions, and cannot be regarded as having such great significance as was formerly attached to it. Convulsions and maniacal excitement are occasionally observed.

The failure of the systemic circulation is evidenced by congestion of the various internal organs, and by œdema of the extremities, but these conditions rarely become so marked as in valvular disease. Albumin may appear in the urine as the result of the disordered renal circulation.

Emboli are occasionally derived from clots formed in the chambers of the heart, and lead to infarction of the internal organs. Repeated epistaxis has sometimes been observed, and when present contributes to the general weakness.

The physical signs of fatty degeneration are rarely distinct. The cardiac impulse is usually weak and diffuse, and when the cavities are dilated the heart-dulness is increased. The cervical veins may be distended, and cyanosis may be present when extreme dilatation has supervened. The sounds of the heart, especially the first sound, are weak, but often remain quite clear. A systolic murmur is frequently audible at the apex. The galop rhythm is heard when dilatation is marked.

Diagnosis of Myocardial Degeneration.—The diagnosis of the various conditions described is always more or less uncertain, though frequently it is possible to make a probable diagnosis.

The occurrence of white infarction or anæmic necrosis may sometimes be suspected when the condition of the peripheral arteries suggests sclerosis of the coronary vessels or when foci from which emboli might take origin have been established, and the patient is suddenly seized with violent angina, asthmatic symptoms, and failure of cardiac power.

Parenchymatous degeneration is, as a rule, so closely associated with acute myocarditis that it is never possible to distinguish them, and the diagnosis is concerned only with the determination of myocardial degeneration or inflammation. The points upon which reliance can generally be placed are weakness of the impulse and pulse and of the first sound of the heart occurring in the course of a febrile disease. In decided cases marked dyspnœa, increase of the cardiac dulness, and embryocardia or galop-rhythm would indicate a high degree of cardiac dilatation secondary to degeneration or myocarditis. In these instances, however, the presence of a systolic murmur at the apex and of decided evidences of weak heart is suggestive of endocarditis or valvular disease, and the influence of time with treatment will be most important in making the distinction.

Fatty infiltration of the heart may be suspected in every obese subject, especially when signs of cardiac weakness are present. In persons not decidedly corpulent the presence of fat about the heart is more difficult to determine, but increase of the area of dulness, with muffling of the sounds and evidences of weak heart, are important factors in the diagnosis, though not distinctive. In women about the menopause syncopal attacks and palpitation of purely nervous origin are quite common, as is also fatty infiltration of the heart with the same symptoms. The determination of the cause of the symptoms is usually easy in such cases, since the physical signs are indicative of a normal heart in the former and of a fatty heart in the latter cases.

Fatty degeneration of the heart is often assumed to be present in cases of valvular disease or of hypertrophy and dilatation when the cardiac power fails, but is in reality quite as often absent as present in these cases. Little value can be attached to the loudness of murmurs, as was pointed out in the article on Valvular Diseases. A harsh murmur may continue after the myocardium degenerates, while a soft murmur may attend an extensive valvular lesion with vigorous muscular walls. When a strong murmur grows progressively more weak and there is also progressive weakness of cardiac action with marked and persistent irregularity, it points strongly to myocardial degeneration, especially of fatty character. Simple fatty degeneration is suspected when the patient has distinct failure of the cardiac power, with weak but clear sounds, syncopal attacks, and angina. The same symptoms, however, present themselves in chronic myocarditis, and the diagnosis is practically impossible. Marked sclerosis of the peripheral arteries, with symptoms of renal contraction and severe anginal paroxysms, would point to the existence of chronic myocarditis, though the muscle might be fatty at the same time. The arcus senilis, to which so much importance has been attached, is by no means a sure indication of fatty degeneration of the heart. In young persons, however, this sign is of some value when taken in combination with other symptoms. Cheyne-Stokes breathing may occur in fatty degeneration of the heart, even when no renal lesion can be demonstrated. But it is not of diagnostic value, since it is much more frequently associated with a uræmic condition.

Prognosis.—Myocardial degeneration of any kind is to be regarded as a serious condition, though the power of the heart may be apparently regained in many cases. Parenchymatous degeneration is frequently followed by complete recovery, but may, on the other hand, lead to fatty degeneration. Fatty infiltration may be temporary, disappearing when general polysarcia is relieved, and under suitable treatment is capable of some relief, unless the muscle-fibres have become extensively degenerated or atrophied. The tendency to sudden death exists in all forms of degeneration, and must be reckoned with in the prognosis. In fatty heart some premonition may be given by the recurrence of syncopal attacks, severe angina, or pseudo-apoplectic seizures, in any of which the patient may suddenly expire. Sudden death not rarely occurs in the course of infectious fevers, such as diphtheria, as a result of extreme myocardial degeneration.

Intercurrent diseases, especially pulmonary and febrile affections, influence the prognosis unfavorably, or, more properly, are themselves of unusual gravity when myocardial degeneration exists.

Treatment.—Anæmic necrosis of the myocardium, if recognized, can only be treated in a symptomatic manner. The anginoid pains may be relieved by the use of nitrite of amyl or by hypodermic injection of morphine, and the heart-power will require stimulation by ether, alcohol, or digitalis. The disease is sudden in its onset, and will therefore demand rapidly-acting stimulants.

Clinical experience has shown the great value of alcohol in the treatment of cardiac weakness in the course of infectious fevers, the pathological condition present being often parenchymatous degeneration. In the same cases strychnine and digitalis may be useful or necessary, but occupy a position second in importance to that of alcohol. The patient must be kept as nearly at rest as may be possible, and if necessary to this end nerve-sedatives or quietants, like bromide of potassium or small doses of opium, may be administered.

Fatty infiltration of the heart is treated in the same way as general polysarcia, such modifications being made as are required by the degree of cardiac weakness. The method of Oertel in these cases is attended by striking results in many instances. It consists in the reduction of the amount of liquid permitted the patient with the food or apart from food, in reduction of the carbohydrates, and finally in graduated exercise. The best results are obtained when the diet is strictly regulated by measure and weight, and the exercise prescribed is gradually increased so as to strengthen the cardiac power. The patient is directed to ascend elevations of various grades and lengths as his heart increases in power. The details of this treatment are, however, more fully considered in the treatment of Obesity.

Medicinal agents probably have little power to reduce the deposited fatty tissue, but the iodide of potassium, in doses of three to five grains three times daily, has seemed to give the best results. When symptoms of cardiac weakness are decided, stimulants will be required. Digitalis should be given with caution on account of the tendency to rupture of the heart, but in small doses is safe and efficient. Nux vomica or strychnine is also of value.

Fatty degeneration of the heart will require a quite different plan of treatment. In this case rest is a most important requisite. The patient must avoid every form of physical or mental excitement which might tax the power of the heart. The diet should be nourishing, but easily assimilated, and but small quantities of food should be taken at a single meal. The general nutrition of the patient is to be improved by the use of tonics, such as arsenic, iron, and quinine or strychnine, and often advantage is derived from residence in the open air during pleasant weather. A little wine may be permitted with the meals, and may contribute to better digestion and nutrition.

When the evidence of failing heart-power becomes marked, the patient must be confined to bed and cardiac stimulants must be administered. Digitalis should be given with the same caution as in fatty infiltration, and is often well combined with the tincture of nux vomica, both remedies being used in small doses. Sudden heart failure requires the prompt effect of the diffusible stimulants, such as ether, ammonium, Hoffmann's anodyne, or alcohol. In cases in which arterio-sclerosis is marked, nitro-glycerin and the nitrites are especially useful.

For the anginoid paroxysms hot applications to the præcordia, whiskey or brandy in hot water or milk, or Hoffmann's anodyne may be useful; or recourse may be had to the more powerful remedies, such as nitrite of amyl and nitro-glycerin. Morphine may be given in small doses, but it must not be used freely in cases in which the cardiac degeneration is marked and the tendency to apoplectiform seizures and coma is evident.

During convalescence from a severe attack of cardiac failure the patient must be cautioned to avoid any unnecessary exertion or mental excitement, and digitalis with iron or other tonics may be employed.

ANEURISM OF THE HEART.

Different portions of the heart are subject to aneurismal dilatation, and it will be necessary to consider separately aneurisms of the heart-wall and of the valves.

ANEURISM OF THE HEART-WALL.—This rare condition, called by the older writers partial aneurism, presents itself as a localized dilatation of a portion of one of the cardiac cavities, and is to be distinguished from general dilatation, to which the term aneurism was formerly applied. It is a very rare disease, and in itself has little clinical importance, though it associates itself with other conditions and sometimes leads to serious results.

Two varieties of aneurism may be distinguished—the chronic and the acute.

Chronic aneurism of the heart-wall is developed slowly at a point which has become inelastic as a result of some myocardial degeneration. Chronic myocarditis or fibroid overgrowth is the almost invariable forerunner of chronic aneurism, though in a few cases fatty degeneration alone has been discovered. In one case, cited in Legg's Bradshaw Lectures, a stab wound of the chest and heart-wall led to a localized aneurismal dilatation, the scar at the point of injury being of course exactly comparable to chronic myocarditis from other causes. Chronic aneurism is more common in men than women, and after middle life than before that period, because the causes which give rise to the condition are more liable to be present in the former than in the latter. In a large proportion of the cases a history of syphilis may be obtained.

Acute aneurism is the rapid dilatation of a point of the heart-wall which has become weakened by acute degenerations or ulcerations. Among these are deep endocardial ulcerations of malignant endocarditis, anæmic softening or myomalacia cordis as a result of embolism or thrombosis of one of the branches of the coronary arteries, and acute myocarditis. Another conceivable cause is the localized weakening which would result from rupture of a myocardial abscess or cyst, but, according to Legg, this has not been shown to have been the cause in any case.

Morbid Anatomy.—Aneurism of the heart-wall in a great majority of all cases affects the left ventricle near the apex and anteriorly, 55 of the 87 cases collected by Pelvet and 59 of those tabulated by Legg having had this situation. In nearly all other cases some other part of the wall of the left ventricle or the septum is involved, but occasionally aneurisms of the auricles or right ventricle have been observed. They vary from dilatations of the size of small nuts to those which are quite as large as the heart itself, or larger. Usually there is but one, though this may be divided into two sacs, as in the case of Janeway, or three pouches may communicate with the one cavity, as in one referred to by Peacock. In a case reported by Thurnam there were four separate aneurisms adjoining each other in the wall of the left ventricle. Usually the opening into the cavity of the heart is large, but instances are sometimes observed in which the aneurism is saccular, communicating with the ventricle by a narrow orifice. The parietal layer of the pericardium is generally more or less attached to the aneurismal sac, and complete obliteration of the pericardial cavity may be observed. In the latter case the aneurism may be completely enveloped by the left lung. Very rarely is the mass of sufficient size to exercise injurious pressure on neighboring structures, but in a remarkable case of Berthold's, an aneurism of the right auricle eroded the ribs and appeared as a pulsating tumor under the skin. The sac of the aneurism is generally more or less filled with laminated clots, which may become almost completely organized and calcareous. Dr. Wilks reported an instance of this kind in which the aneurism was practically cured. In chronic aneurisms all three layers of the heart-wall, the endocardium, myocardium, and pericardium, are generally retained in the walls, but are thickened, and frequently the muscular fibres of the middle coat are largely replaced by dense fibrous tissue. The wall may be still further altered by calcareous infiltration. Rupture of the sac is a rare event, having occurred in but 7 of Legg's 90 cases. Acute aneurisms are more prone to rupture, since the wall is so much weakened by degeneration, and, in cases of ulcerative endocarditis, by destruction of the endocardium and portions of the myocardium. Aneurisms of the septum project into the right ventricle on account of the greater pressure on the left side, but if developed during fœtal life the reverse conditions obtain, as was seen in a case of Rokitansky's.

Among the lesions associated with cardiac aneurism are those which result from the same fundamental causes. Arterio-sclerosis is generally present, and valvular lesions are not infrequent. When the power of the heart fails, the internal organs may be highly congested. Pericardial adhesions may be secondary, or may contribute to the formation of aneurism, by occasioning localized degeneration of the heart-wall at their attachment.

The **symptoms** of cardiac aneurism are never distinctive, and there may be no indication at all of a cardiac lesion. Usually the symptoms of fibroid degeneration or fatty heart are prominent, and the aneurism remains unsuspected. In one of the most marked cases which have come under my observation the physical signs and symptoms of aortic regurgitation were unequivocal, but at autopsy, in addition to the valvular lesion, there was found an aneurism of the left ventricle as large as an apple.

On physical examination a localized enlargement of the heart may be detected, but is not distinguishable from hypertrophy and dilatation of one of the cavities. Superficial pulsation was observed in Berthold's case, but is extremely rare. In one of my cases, the aneurism, springing from the left ventricle, was of large size. The heart was displaced so that its impulse was midway between right nipple line and sternum. When the patient was first seen, there was very extensive left-sided pleural effusion. In proceeding to aspirate this in the sixth interspace on the anterior line of the left axilla the trocar entered the aneurism of the heart. So far from doing harm the withdrawal of eight ounces of blood was followed by marked relief, the pleural effusion was absorbed, and then, while the heart was still to the right of the sternum, the large cardiac aneurism could be felt pulsating strongly in the fifth left interspace, outside of the nipple line. In case of rupture of the aneurism the sympoms of rupture of the heart are noted.

The **diagnosis** of aneurism of the heart is rarely possible. If a localized enlargement were developed under observation in a case of chronic myocarditis, aneurism might be suspected, but cardiac dilatation would more frequently be found post-mortem. Large aneurisms causing superficial pulsation are indistinguishable from aneurisms of the aorta or other vessels unless, as in the case reported above, the point of pulsation and its relations with the cardiac impulse calls attention to the possibility of this rare lesion.

The **prognosis** is extremely grave. Sudden death from syncope and rupture occasionally results, but more commonly death is gradual and due to cardiac exhaustion from chronic myocarditis or fatty degeneration rather than from the aneurism itself. The rare possibility of obliteration of the sac by laminated clots has been referred to.

The **treatment** is simply that of fibroid or fatty heart.

Aneurisms of the Heart-valves are occasionally found in cases of ulcerative endocarditis, when one of the endocardial reflections of the valve has been destroyed. They appear as spherical projections upon the segments, the concavity facing the greater blood-pressure. Thus in the aortic valves the aneurism bulges into the left ventricle; and in the case of the mitral valve, toward the left auricle. The aortic valves are most frequently affected. Of the mitral segments, the anterior is more commonly involved than the posterior. Sometimes several pouches are found in the same segments. Rupture of the aneurism may occur, and leads to serious valvular incompetency. A form of chronic valvular aneurism is observed in cases of chronic sclerotic valvulitis, in which the whole segment gradually yields to the intracardial or aortic blood-pressure. In the case of the bicuspid valves dilatation of the sinuses of Valsalva may coexist. Gradual pouching of the anterior mitral leaflet is sometimes observed in cases of aortic valve disease.

Valvular aneurisms give rise to no peculiar symptoms nor can they be diagnosticated, unless rupture occurs, when the sudden development of failure of the valve would indicate rupture of a valvular aneurism or detachment of one of the segments.

RUPTURE OF THE HEART.

This condition is rare and has little practical importance, excepting that it is one of the causes of sudden death.

Etiology.—In all cases some degenerated condition of the myocardium weakens the wall of the heart and leads to the rupture. Fatty degeneration and infiltration are the most frequent lesions observed. Of 100 cases collected by Quain, fatty degeneration was detected in 77, but according to his own description the lesion in some of these cases was rather anæmic necrosis following thrombosis of the coronary arteries. Uniform fatty degeneration is less liable to lead to rupture than are localized areas.

Circumscribed myocarditis, softening of the heart as a result of thrombosis or embolism of the coronary vessels, degenerated tumors or gummata, and deep endocardial ulcerations, may all occasion rupture. Richard recorded an instance in which the perforation occurred at a point where the myocardium was atrophied and weakened by the pressure of an aneurism of one of the coronary arteries. Rupture of a true cardiac aneurism is a rare event.

Rupture of the heart is rarely observed in young persons: two-thirds of Quain's cases were over sixty years of age. It is slightly more common in the male than in the female sex.

The determining cause may be strain, though in some cases the patient has been at perfect rest at the time of rupture. Lifting heavy weights, straining at stool, violent emotional excitement, coughing, and vomiting are among the causes which are generally noted.

Morbid Anatomy.—The perforation is found in the left ventricle in a great majority of the cases, and generally in the anterior wall near the apex and parallel with the septum. More rarely the posterior wall of the left ventricle, the septum, or the wall of the right ventricle is torn. The external opening may be scarcely visible, or may be an inch or more in length. The rent is usually parallel with the muscle-fibres, though rarely the latter may be ruptured transversely. The pericardial opening is usually the smaller, but occasionally the larger; and sometimes a small internal opening communicates with a number of rents on the pericardial surface. The communication between the openings may be oblique or quite sinuous.

The pericardial cavity is more or less filled with blood, sometimes several pounds being found post-mortem. If, however, the pericardial layers are united by adhesions, the rupture may communicate with the pleural cavity, and this may contain large quantities of clotted blood. The rent in the cardiac wall is usually closed by a clot, and the muscle is infiltrated with blood near the edges of the rupture. Microscopically, there is evidence of some form of cardiac degeneration which has led to the perforation, and also secondary fragmentation of the adjacent fibres.

Partial ruptures are sometimes found associated with the complete tear, but may occur independently. A rare accident is rupture of one of the papillary muscles or of a valve-segment. More frequently one of the chordæ tendineæ becomes detached.

Symptomatology.—The symptoms of cardiac rupture come on with great suddenness, and the patient may die in a few moments. Nearly always there is sharp pain at the heart, and frequently this is agonizing in its severity. The patient at once grows pale, breaks into profuse perspiration, and may fall heavily to the ground. There is always profound terror or apprehension, the surface of the body is cold, the pulse extremely rapid and weak; dark spots are seen before the eyes, and there is a roaring sound in the ears. Very soon the patient falls into syncope. Not rarely profuse vomiting and purging are noted, and the appearance of cholera may be closely simulated.

A majority of all cases die suddenly, but in rare instances life may be preserved for several days, and especially is this the case when the septum alone is ruptured. In a case reported by Mays the patient lived for seventeen days after the occurrence of the rupture.

Physical Signs.—If considerable blood has accumulated in the pericardial cavity, the disappearance of the apex-beat, the weakness and muffled character of the heart-sounds, and the increase of the dulness on percussion are significant symptoms. When, however, little blood has escaped, the sounds may be quite clear and loud.

Partial rupture, when it causes detachment of one of the papillary muscles, chordæ tendineæ, or valve-segments, causes more or less sharp pain and rapidly increasing evidences of cardiac failure. The patient may suffer extreme dyspnœa and become cyanosed to the last degree. The suddenness of the attack and the violence of the symptoms are often most alarming; and indeed sudden death may readily occur. On physical examination the cavities of the heart may be found enormously dilated, and murmurs of valvular incompetence are often present. In one of my own cases the symptoms were those above named, and the murmur, a loud whirring sound, was reported to have at once become audible at a distance from the patient. If the valvular inadequacy be not extreme, secondary hypertrophy and dilatation of the heart may develop, and compensation may be thus established. Even after marked temporary improvement, I have, however, known a sudden recurrence of all the symptoms of heart failure to occur with speedy death.

Diagnosis.—In cases of sudden and severe cardiac pain, followed by fainting and other evidences of internal hæmorrhage, rupture of the heart may always be suspected. Its occurrence cannot, however, in many cases be certainly diagnosticated. Muffling of the heart-sounds and increase of the præcordial dulness are important signs, and may, with the symptoms present, lead to a complete diagnosis.

The existence of partial cardiac rupture also cannot usually be determined positively. Rupture of a segment of a valve may, however, be diagnosed with some probability in cases such as my own above referred to.

Prognosis.—Complete rupture of the heart is always fatal. In some cases fibrous overgrowth has been found at the point of rupture, and has been regarded as evidence of a reparative tendency. On the contrary, such fibroid areas are merely a part of the degenerative lesions which occasion rupture.

Death may occur very suddenly from anæmia of the brain or from what we must call heart-shock, in which the heart suddenly ceases to beat without the external pressure of large accumulation of blood in the pericardial cavity, and without sufficient hæmorrhage to cause anæmia of the brain. In cases of more gradual death the pressure of the blood in the pericardial sac soon arrests the cardiac power.

Treatment.—Prophylaxis is the most important part of the treatment. Persons known to have degenerative lesions of the myocardium should avoid all forms of mental or bodily strain. They must lead quiet and tranquil lives, regulating their habits and work in a way to promote this end. Constipation may be obviated by dietary measures or by the use of salines. If constipation does occur, enemata may be employed to prevent excessive straining at stool.

When rupture has occurred, the patient must be put at complete rest and morphine should be given freely. An ice-bag over the heart or hot applications may give relief, and tincture of aconite may be cautiously administered to quiet overaction. Stimulants of all kinds must be avoided unless the heart is evidently failing, when whiskey or brandy and digitalis may be given. Under any circumstances these remedies are apt to increase the hæmorrhage. Ergot and turpentine have no power to stop the loss of blood, and their employment is worse than useless, since they tend to increase blood-pressure.

When the symptoms indicate the occurrence of rupture, and when death does not occur almost instantly, the hope may be entertained that the rupture is but partial. The patient must be put at complete rest and morphine should be given hypodermically in doses sufficient to allay urgent distress. An ice-bag over the heart or hot applictions may further aid in giving relief. So much shock is often present that powerful diffusible stimuli must be administered, and it may be necessary to use ether and strychnine hypodermically in full doses. Hæmoptysis is not rare and astringents are of no avail, or indeed, as in the case of ergot or turpentine which tend to increase blood pressure, they may be injurious. If reaction is secured, absolute rest must still be insisted upon for a considerable period. Cardiac sedatives may now be required to control overaction. The diet must be carefully restricted. The occasional application of dry cups around the base of the chest may be indicated; or it may even be desirable to withdraw small amounts of blood by wet cups or by cautious venesection. Any return to exertion must for a long time at least be most guarded.

NEW-GROWTHS AND PARASITES OF THE HEART.

New-growths of the heart may originate in the myocardium or endocardium, or may invade the organ from the pericardial surface. Primary cancer and sarcoma have been observed, but are exceedingly rare. Secondary malignant deposits are much more frequent. Usually they occur as small nodular masses situated in the muscular substance and projecting somewhat from the surface. There may be but one or two, or the myocardium may be thickly studded, as was the case in a recent instance under my observation. The heart may be secondarily involved by sarcomatous growths of the mediastinum. In these cases the pericardial reflections are first invaded, and from these the process extends to the cardiac walls. Myxomatous, lipomatous, and fibrous tumors are occasionally observed, while nodular myomata and cystic tumors are extremely rare. The latter may be single or multiple, and often contain fluid tinged with blood. Cases have been reported in which the myocardium presented innumerable small cystic formations containing clear fluid.

Myxomatous, chondromatous, and sarcomatous new-growths of the endocardium may be met with, but are exceedingly infrequent. Polypoid tumors of the auricles and of the ventricles near the apex have been reported, but there is always considerable doubt whether such be not transformed cardiac thrombi. In a few instances, however, unquestionably there have been real polypoid tumors.

Syphilitic gummata of the heart are most frequent in the left ventricle, but may occur in any part of the organ. Distinct nodular growths or diffuse syphilitic infiltration leading to fibrous myocarditis may be observed, the latter, however, being far more common. Miliary tubercles and caseous tubercular masses are sometimes seen in the pericardium and invading the muscular substance.

Hydatid cysts, cysticercus cellulosæ, and the larval pentastomum denticulatum are rare forms of parasitic disease of the heart. The hydatid cysts are more frequently observed in the right ventricle than elsewhere, and vary in size from that of a pinhead to that of a small apple. Rupture, either internally or into the pericardium, is not uncommon, having occurred in 6 of 21 cases. In most cases hydatid cysts of the liver or other organs are present at the same time, and if rupture occurs into the right ventricle, secondary cysts of the lungs may be developed.

The **symptoms** of tumor of the heart are never distinctive. In some cases indefinite indications of cardiac weakness are present, but in many the condition is unsuspected until it is discovered at autopsy. Endocardial tumors may cause obstruction of the valvular orifices, especially when, as in the cases of Gairdner and of Jürgens, they are polypoid in nature. Degenerated gummata or cysts may occasion cardiac rupture and sudden death. Embolic manifestations are sometimes noted when portions of the new-growth within the heart are broken off and carried into the circulation.

The **diagnosis** can never be made, and the **prognosis** therefore requires no consideration.

In syphilitic cases the iodide of potassium in large doses would probably exercise the same happy influence as in the case of gummata or syphilitic infiltration elsewhere.

NEUROSES OF THE HEART.

BY WILLIAM PEPPER.

UNDER this head are included several functional affections of the heart more or less dependent on disturbances of the innervation.

PALPITATION.

Palpitation is the subjective sensation of forcible or rapid action of the heart. As a rule, the rate and force of the heart's action are considerably increased, but it is not at all necessary that this should be the case; and, on the other hand, the heart may be beating very rapidly, though the patient is entirely unaware of any disturbance.

Etiology.—The occurrence of palpitation always suggests to the patient and the physician the idea of valvular or other cardiac disease, but in many cases, perhaps in the majority, other causal elements will be discovered. Valvular disease, hypertrophy and dilatation of the heart, and myocardial affections must, however, be suspected in every case as important and serious causes.

The general condition of the patient has much to do with the occurrence of palpitation in some cases, and particularly do anæmia, chlorosis, hysteria, and neurasthenia play an important part. Attacks of palpitation are not uncommon in young girls or boys about the period of puberty. Some women suffer habitually with palpitation during menstruation, and it is one of the distressing incidents of the menopause. Hysteria and neurasthenia are important predisposing causes, and patients convalescing from serious illness, particularly the fevers, are liable to attacks on slight provocation. In most of these conditions a lowered state of the arterial tension plays a part as one of the etiological factors.

The attack is often occasioned by mental excitement, especially by fright, or by physical exertion. Gastric disorder so often serves to excite or to aggravate palpitation that it demands special consideration. Sometimes a particular article of diet, as strawberries or shellfish, cannot be eaten without a violent attack, though the patient is unaffected by mental emotion or exertion. In other cases the reflex irritation from the catarrhal gastric membrane maintains almost constant over-action of the heart; and this is increased to severe palpitation after nearly every meal. Tea, coffee, and alcohol are frequent causes, both by their action on the nervous system and by their tendency to derange the stomach. Excessive smoking is of equal importance in many cases. It is obvious that various disturbances of the ureters, ovaries, or other organs may excite palpitation by reflex irritation.

Symptomatology.—Palpitation is essentially a paroxysmal affection, coming on, as a rule, with great suddenness, though the patient often experiences some vague sense of foreboding and becomes pallid before the attack begins. The important symptom is the sensation of violent cardiac action. Generally, this is accompanied or dependent on actual increase of force and rapidity of the heart, but not necessarily. The impulse is strong, diffuse, and plainly visible. The superficial vessels throb, and the whole surface becomes somewhat flushed. In extreme cases the sense of cardiac beating and general vascular throbbing becomes extremely distressing. The pulse is strong and full, and may be greatly increased in frequency : a rate of 120 to 150 is not unusual. More or less dyspnœa accompanies the increase of cardiac rapidity.

The attack may subside gradually or suddenly, and the patient is generally more or less exhausted if the paroxysm has been prolonged. The whole attack may last but a few minutes or it may continue for hours or days.

On physical examination some enlargement of the heart is often discovered, the impulse is found extremely forcible, and the sounds are louder than normal.

Diagnosis.—Purely nervous palpitation must be distinguished from that of valvular disease. As a rule, the absence of cardiac murmurs during the intervals between attacks and the absence of hypertrophy lead to a correct diagnosis. When anæmic murmurs are present, it is extremely difficult to arrive at a positive diagnosis, but a careful examination of the patient's general condition will do much to determine the cause of the palpitation.

Prognosis.—Palpitation in itself is not usually serious, though in elderly persons with brittle arteries cerebral hæmorrhage may occur during an attack. Repeated palpitation may lead to some hypertrophy.

Treatment.—The treatment of palpitation will be considered with that of Tachycardia.

TACHYCARDIA.

SYNONYMS.—Paroxysmal Hurry of the Heart; Synchosphyxia; Rapid Heart.

Definition.—The term "tachycardia," in the strictest sense, means rapid action of the heart from any cause, but it has been applied more particularly to cases of paroxysmal rapidity of the heart. Tachycardia is distinguished from palpitation by the absence of subjective sensations of forcible action, but in reality tachycardia and palpitation are so frequently combined that a rigid separation is impossible. In some cases of tachycardia with inordinate rapidity of the heart there is absolutely no sense of palpitation, whereas in other cases subjective sensations are pronounced.

Etiology.—Excessive rapidity of the heart is occasionally noted as a physiological condition in certain individuals, and is not unfrequently present in women during gestation. Some individuals, indeed, have the power to increase the rate of the heart at will, as in the case of a student of Tarchanoff's, who was able to increase the pulse-rate from 70 to 120.

Pathological rapidity of the heart may be divided into two groups of cases—those which are symptomatic of some other condition, and those which are essential or which are more strictly of the nature of a neurosis. In either case, however, the cardiac innervation is in some way affected, and certain authors have sought to distinguish cases due to stimulation of the sympathetic nerve and cardiac ganglia from those which result from paresis of the vagus (Bamberger, Rommelaere, Nothnagel). Such a distinction is occasionally profitable in a clinical sense, but cannot, as yet, be followed out in all cases.

Symptomatic tachycardia may be due to certain general conditions which affect the nervous excitability, to lesions involving the cardiac nervous mechanism, centrally or peripherally, or to reflex action in organic affections of various organs.

Abnormal irritability of the nervous system, such as is met with in neurasthenic individuals or young persons who have grown rapidly and are weakened and anæmic, frequently occasions rapid action of the heart under the influence of trivial causes. Excessive venery, masturbation, mental overwork and fatigue, and spinal irritation act in a similar manner. Tachycardia is sometimes marked in young women suffering with chlorosis and in persons who have become anæmic by loss of blood or otherwise. In the same class of causes must be reckoned toxic influences, of which the excessive use of tea, coffee, tobacco, and alcohol are frequent examples. In these cases also gastric or other visceral disturbances frequently add to the derangement of the heart by reflex action. The rapidity of the pulse in fever is one of the most familiar facts in medicine, though the cause remains to a certain extent obscure. Sometimes the pulse becomes of inordinate rapidity, as in scarlet fever and diphtheria, and there may be persistent increase of the pulse-rate during convalescence, and even, as in the case reported by Pribram, for years after the disease.

The lesions which induce tachycardia by involvement of the nervous mechanism affect the central or peripheral portions of the vagus or sympathetic. Tumors, hæmorrhage, or softening of the medulla or cord occasionally give rise to great rapidity of the pulse, which may continue to the end of the patient's life. Peripherally, tumors or aneurisms may press upon the vagus in the neck or thorax. A frequent cause is overgrowth of the tracheal and bronchial lymphatic glands. In these cases the pressure alone may suffice to paralyze the vagus, or there may be associated neuritis. In cases of polyneuritis the vagus or sympathetic of the neck may be involved with the other nerves.

One of the most decided paroxysms of tachycardia I have ever seen occurred a few hours before death in a woman suffering with general alcoholic neuritis, and, from the associated symptoms, was evidently due to implication of the sympathetic nerve. In cases of locomotor ataxia there is almost habitually some increase of the pulse-rate, and occasionally attacks of great tachycardia are observed. In one of my cases the rate remained about 235 during an attack lasting several hours. How much peripheral neuritis has to do with the rapid heart of this disease remains to be determined. According to Charcot, the rate is increased to an habitual 90 to 100. Spender of Bath and Sansom have called attention to the tachycardia which accompanies and sometimes remains after osteo-arthritis.

The reflex causes of rapid heart are exceedingly numerous, and in this group may be counted lesions of the heart and vascular system, whose influence is probably reflex. Tachycardia is at times noted in myocarditis, in valvular disease, especially mitral, in affections of the aorta, and associated with angina pectoris in various pathological conditions of the heart and arterial system. Digestive disturbances are common exciting causes, or may be the only assignable etiological factor in certain cases. In a remarkable instance, occurring in a patient with gastritis, I saw a paroxysm which had lasted fifteen hours, and during which the pulse varied from 220 to 240 per minute, speedily subside after administration of alkaline remedies and eructation of gas. Intestinal irritation is more commonly operative in children, and particularly that due to intestinal worms. In females, at the establishment of menstruation and in the climacteric, uterine and ovarian disorders are frequent causes of paroxysms of tachycardia. Occasionally nasal hypertrophies, eye-strain, and diseases of the mouth cause reflex excitement of the cardiac action.

Finally, there is the group of cases which are generally designated as essential tachycardia or paroxysmal tachycardia, and whose nature seems to be that of a neurosis of the heart. In some of these the patient presents no abnormal condition, excepting the attacks of tachycardia; in others he is the subject of some other neurosis with which the rapid heart is associated. Various disturbances of the general health or of particular organs may determine the attack in such cases, but the fundamental cause of the disease remains obscure. Paroxysms of this nature occur in exophthalmic goitre, in epilepsy, and in hysteria. In the first-named disease the heart is usually continuously rapid, and occasionally subject to attacks of great hurry. The essential paroxysmal tachycardia, in which the cardiac rapidity is due to an independent neurosis, has been considered by some authors (Trousseau; Sansom) as a form of masked or abortive Graves' disease, the other symptoms failing to develop. Some color is lent to this opinion by the fact that in some cases of exophthalmic goitre paroxysmal hurry of the heart precedes the other symptoms by a long time.

The "irritable heart" of soldiers, described by DaCosta, belongs to the same group of cases, though here the influence of anxiety, over-strain, and illness contributes to the development of cardiac excitability. In a study of many cases DaCosta found the pulse-rate increased out of proportion to the respiration, especially after exertion; sometimes præcordial pain, and occasionally hypertrophy when the disease had lasted a considerable length of time.

Morbid Anatomy.—Few opportunities for post-mortem examination present themselves, and no characteristic lesions have been discovered. In certain cases of typical paroxysmal tachycardia myocardial degeneration, fibroid or fatty, was discovered. Neuritis of the vagus has also been observed.

Symptomatology.—There are two distinct classes of cases—that in which the rapidity of the heart is permanent, and that in which paroxysmal attacks are observed. It is to the latter class especially that the term "tachycardia" is applied, and it is in cases of this class that the symptoms are most pronounced.

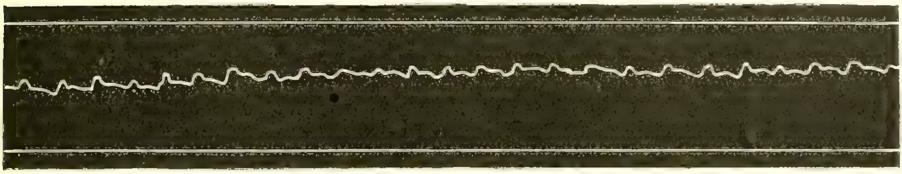
The attack of tachycardia, as a rule, comes on with great suddenness, and sometimes entirely without warning to the patient. In other cases vertigo, ringing in the ears, or a vague sense of foreboding may give an indication of the approaching paroxysm. The attack may last but a few minutes or may continue for hours or days. During its continuance the great rapidity of the pulse is the striking symptom. A rate of 200 is not unusual, and it has been known to reach 300 beats per minute. The pulse is small, weak, easily compressible, and frequently irregular. Very often, in cases of great rapidity, some of the cardiac contractions fail to give rise to a corresponding pulse-beat, and we must therefore count the heart-beats by auscultation rather than the pulse. The patient is at the onset more or less pallid, but during the attack becomes flushed, and may be distinctly florid. The respirations are generally increased, though not in the same proportion as the pulse. Sometimes, however, the respirations are perfectly quiet and natural when the pulse-rate exceeds 200. This was the case in two of my patients. Usually there is dyspnœa, and the rate of respiration is generally from 30 to 40. The absence of respiratory symptoms in certain cases was taken by some observers as an indication of stimulation of the sympathetic nerve rather than involvement of the vagus.

Subjective symptoms are frequently present. Usually there is a feeling of constriction at the heart or general smothering, and, rarely, the sensation amounts to actual pain. Roaring in the ears, hemicrania, vertigo, and a tendency to syncope may persist throughout the attack. The sense of palpitation is sometimes completely absent, and, indeed, the patient may feel as if the heart were slowed or about to stop its beating when the rate is inordinately rapid. In many cases, however, the sense of palpitation coexists with tachycardia. The patient's face gives evidence of the painful nature of the subjective sensations. The voice is generally weak, and may sink to a whisper. Hysterical symptoms are frequently present.

Examination of the heart shows great increase in the rapidity of the contractions. The impulse is diffuse, forcible, and generally irregular, and the heart may be slightly enlarged. The first sound is clear and ringing, but the second, as heard in the second right intercostal space, may be exceedingly weak, from the fact that a smaller quantity of blood is expelled at each systole than is normally the case. The second pulmonic sound may be accentuated. A systolic murmur is sometimes heard at the apex. The long pause between the diastole and succeeding systole of the ventricle is sometimes shortened, and the cardiac rhythm becomes of fœtal type—the condition described as embryocardia.

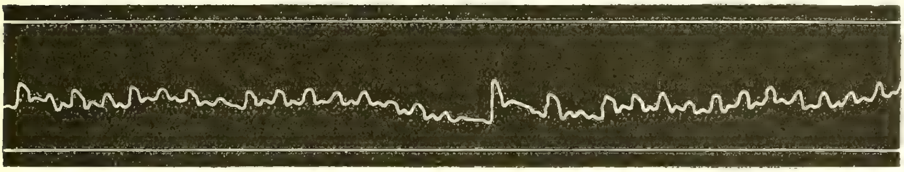
The carotid vessels throb visibly, and a murmur is heard in them on auscultation. Pulsation of the veins may occur toward the end of the attack. The sphygmogram in tachycardia is not distinctive, but the extreme frequency of the pulse is seen in the number of waves in a given space. Sometimes the ordinary characters of the tracing are completely effaced, and it becomes monocrotic; in other cases distinct dicrotism is observed. The latter is an evidence of the low arterial tension which has been regarded by some authors as an important factor in producing tachycardia. (See Figs. 18 and 19.)

FIG. 18.



Sphygmogram from a case of Parenchymatous Nephritis during an attack of Tachycardia. Pulse-rate, 175.

FIG. 19.



Sphygmogram from a case of Phthisis, during an attack of Tachycardia. Pulse-rate, 184. Low arterial tension; dicrotism.

If the attack continues long, more or less congestion of the viscera results. The liver may become enlarged, the urine scant, and cough and increase of dyspnœa may denote impaired pulmonary circulation. Vertigo, insomnia, and delirium are indicative of disturbances of the cerebral circulation.

The termination of the attack may be as sudden as the onset, and may be attended by eructations or vomiting. In other cases the heart-action gradually grows less frequent until the normal is reached.

Diagnosis.—The distinction between tachycardia and palpitation has been referred to, but in a majority of the cases this distinction cannot be rigidly maintained. The sense of palpitation present in paroxysms of tachycardia is, however, relatively far less pronounced than in cases of ordinary palpitation, while the pulse-rate is much greater.

Essential tachycardia must be distinguished from the symptomatic forms. Valvular disease may be excluded by careful auscultation of the heart. Myocardial disease is less readily detected, but there may be important indications in the intervals between the attacks of rapid action, and the etiological features may point to myocarditis.

Essential tachycardia may be found to depend upon neurasthenia, hysteria, or obscure exophthalmic goitre, and each of these conditions must therefore be considered in the final diagnosis.

Prognosis.—In itself, tachycardia rarely leads to serious consequences. In old persons cerebral hæmorrhage might occur at the onset of the paroxysm. Repeated or long-continued attacks sometimes give rise to hypertrophy of the heart. Sudden death from heart failure occurred in four of the series of cases of paroxysmal tachycardia analyzed by Bouveret. In a case of myelitis of the cervical cord I saw tachycardia suddenly develop, and in a few hours the patient succumbed, apparently from heart failure. At the autopsy the cord showed numerous small hæmorrhages in the affected area. The prognosis, as far as cure is concerned, is unfavorable, and many patients grow discouraged and despondent, thereby adding to the gravity of the case. On the other hand, I have under observation patients who have for many years been subject to sudden and violent paroxysms, apparently due to some derangement of the nervous mechanism of the heart, and who in the intervals enjoy excellent health, and are exceptionally active in habit. Symptomatic tachycardia is generally relieved when the primary disease is removed, but may remain as a permanent condition. Either the symptomatic or the essential form may, as above stated, continue for many years without disturbing the general health.

Treatment of Palpitation and Tachycardia.—Prophylactic measures are often of great service in preventing attacks, and will be suggested in individual cases by a careful study of the patient. In some instances particular articles of diet, flatulence, constipation, or other slight causes invariably provoke an attack. The avoidance or removal of such causes will therefore be the first point in the treatment.

During the paroxysm the patient must be placed at absolute rest in bed and in a darkened room. Sometimes the paroxysm may be cut short by firm pressure over the vagus and sympathetic in the neck or in the region of the ovaries. In other cases the use of the electric brush in the præcordial region has been attended with happy results. An ice-bag placed over the heart is probably the most uniformly satisfactory form of local treatment, and may be supplemented by a draught of cold water or by swallowing pieces of ice.

Internally, a large number of remedies have been employed, and trial alone will determine which will act most happily in a given case. Sometimes a cup of strong black coffee will immediately check the paroxysm; in other cases bromide of potassium, valerianate of ammonium or zinc, asafœtida, or camphor may be more serviceable. The most certain and powerful remedy, but one which is generally to be held in reserve or used with caution, is morphine. The best results are obtained from the hypodermic injection of morphine (gr. $\frac{1}{12}$ to gr. $\frac{1}{6}$) with atropine (gr. $\frac{1}{150}$). In cases in which a tonic was desirable in addition to the nerve-sedative, I have found the following suppository of value:

℞. Quininæ sulphatis,	ʒss ;
Ext. opii,	gr. iij vel gr. vj ;
Asafœtidæ,	gr. xviij vel gr. xxx ;
Ol. theobrom.,	q. s.—M.

Ft. suppositoria No. vj.

The use of inhalations of chloroform or nitrite of amyl has sometimes been advised, but is to be discouraged.

During the interval between the paroxysms the general health of the patient is to be studied with care, and suitable general hygienic and dietetic regulations instituted. Tea, coffee, and tobacco should be discontinued, and alcohol but sparingly used, if at all. Anæmia and chlorosis may require the administration of iron and arsenic, and general tonics are frequently of value. Local treatment may be required by gastric disturbances, intestinal parasites, or uterine and ovarian disease.

For the cardiac condition itself digitalis in moderate doses is most serviceable, and may be used continuously for long periods of time. Its action may be explained in some cases by its power to raise blood-pressure, a lowered state of the arterial tension probably having some influence in the occurrence of tachycardia and palpitation. Ergot, arsenic, and nux vomica are also of value in certain cases.

Galvanism of the vagus and sympathetic has sometimes acted favorably. The current should be weak and descending, and may be applied for a few moments every day or every other day.

BRADYCARDIA.

Bradycardia and brachycardia are terms applied to slow action of the heart, either permanent or paroxysmal.

Etiology.—Cases of slow heart may be divided into those which are physiological, those symptomatic of some other affection, and a small group in which the bradycardia is of the nature of a neurosis.

It is said that certain individuals have normally slow-acting hearts, and that the tendency may be inherited; but the question always arises whether there is not some underlying disease in such cases. A patient of my own told me his pulse had always been slow and that physicians had commented on it. He now has aortic stenosis, and this may have been overlooked in former years. The remarkable case cited by Broadbent of an athletic young man, a foot-ball player, whose pulse was 36, is also in point. His sudden death, while swimming, of "cramp" would suggest an obscure cardiac disease.

Bradycardia is commonly noted in women immediately after childbirth. In about 25 per cent. of women during the puerperal period Blot found a transient fall of the rate to 40 per minute.

A similar slowing of the pulse is noted after some of the acute fevers, and has been referred by Traube to cardiac exhaustion. It is most frequently observed after typhoid fever, diphtheria, pneumonia, erysipelas, and acute rheumatism, and may sometimes be quite marked, as in the instance cited by Baumgarten in which the pulse sank to 25 late in a case of diphtheria. Riegel analyzed 1047 cases in which the pulse-rate was below 60, and of these found a greater number resulting from acute fevers than from any other cause. Disease of the digestive tract stood next in point of frequency, 379 of the cases having some gastro-intestinal disturbance as the underlying cause. Chronic dyspepsia, gastric ulcer, cancer of the œsophagus or stomach, and diseases of the liver were all noted in this group. In cases of jaundice the intoxication of the blood is the direct cause of bradycardia. Disease of the urinary organs is an occasional cause, and especially when uræmia supervenes, the slowness of the heart in the latter case doubtless resulting from toxic agents, as in jaundice. Emphysema of the lungs and other pulmonary affections are sometimes observed.

Bradycardia as a symptom of cardiac disease is most frequently met with in cases of coronary sclerosis and myocardial degeneration, particularly fatty and fibroid. In such cases the pulse-rate is often as low as 40 or 50, and has been noted at 12. Slow heart is unusual in valvular disease without myocardial complication, but occurs more frequently in aortic stenosis than in any other form.

Lesions of the nervous system sometimes induce slowness of the heart by direct involvement of the cardiac nervous mechanism or by reflex action. The symptom has been noted in the early stage of meningitis, in apoplexy, in tumors of the cerebrum or medulla, and in injuries of the cervical cord. Czermak was able to arrest his own heart for several beats by pressure of the vagus against a bony outgrowth from one of his cervical vertebræ, and Concato observed the same thing in a patient.

Among the toxic agents which sometimes induce slowing of the pulse are tea, coffee, alcohol, and lead. The first three, however, more frequently induce rapidity or arrhythmia. The poisons of uræmia and jaundice are well-known causes.

The general condition of the patient contributes in some cases or may alone produce retardation of the pulse, as is seen in occasional cases of chlorosis, marked anæmia, diabetes, profound physical exhaustion, and a large number of diseases.

Finally, there is a group of cases in which bradycardia is associated with a neurosis or is itself of this nature. In epilepsy there is sometimes very marked slowing of the pulse, and in a majority of these cases more or less arrhythmia is associated. In one case the rate was reduced to 7 beats per minute (Jacobi), and rates of 25 to 40 have frequently been observed. Bradycardia is also associated with certain cases of hysteria, of mania, and of general paresis. Finally, paroxysms of slow heart may occur without any other morbid symptom, or they may follow fright or other nervous excitement or severe pain.

The physiological cause of the slowing of the pulse probably varies in different cases. In some, as those cases following fevers or exhausting physical exercises weakness of the cardiac muscle or intracardial nervous ganglia probably plays an important part. In others irritations of the vagus, centrally or in its peripheral portions, seems to be the etiological factor concerned. Reflex stimulation of the pneumogastric centre is probably present in the instances of bradycardia which result from diseases of the digestive tract.

Symptomatology.—The important symptom is the slow action of the heart. This may be constant, and give rise to no other symptoms, or may come on in paroxsyms, in which the patient becomes faint or even completely unconscious. Sometimes twitchings of the muscles or epileptiform convulsions are associated, but in case of the latter it is probable that the bradycardia is merely a symptom of epilepsy. These paroxysms may come on without warning, or may follow certain premonitory symptoms, such as vertigo, roaring in the ears, or the sense of impending death. The onset is generally sudden, and the return to the normal condition is similarly abrupt, though a gradual termination is more frequent than a gradual onset. The pulse is weak, small, and slow; but it is always necessary to compare the rapidity of the heart as determined by auscultation, with the rapidity of the pulse, since some of the beats may be so weak that they give rise to no pulse at the wrist. Sometimes every other beat fails to produce a pulse-wave, and in such the heart-action is just twice as frequent as the pulse-rate. The number of pulsations per minute is often remarkably low. A rate of 20 or 30 has often been observed, and several are recorded in which the pulse was but 7 or 8 per minute. The impulse of the heart is weak and the sounds may be scarcely audible. The sphygmographic tracing of the pulse is not distinctive, but, as a rule, the upstroke is slanting, the curve only moderately elevated, and the first wave markedly prolonged. Repeated pulsations on the down stroke are commonly observed.

Diagnosis.—The slowness of the pulse is always a distinctive symptom if by auscultation each ventricular contraction is found to give rise to a pulse-beat.

Prognosis.—Bradycardia in itself is not a serious condition, but when associated with myocardial disease or cerebral affections the prognosis is extremely grave. Sudden death is not unusual.

Treatment.—Cardiac and general stimulants are always of service. In cases in which the arterial tension is high, nitro-glycerin or nitrite of sodium may be used with advantage. In other cases alcoholic stimulants, strychnine, and ammonia are of value. Digitalis tends to slow the pulse still more, and should therefore be avoided. In some cases, however, in which the power of the heart is distinctly lessened, this remedy is required, and acts most happily in small doses. It should only be used when the patient is under constant observation.

ARRHYTHMIA.

SYNONYMS.—Allorhythmia; Delirium cordis; Irregularity of the pulse.

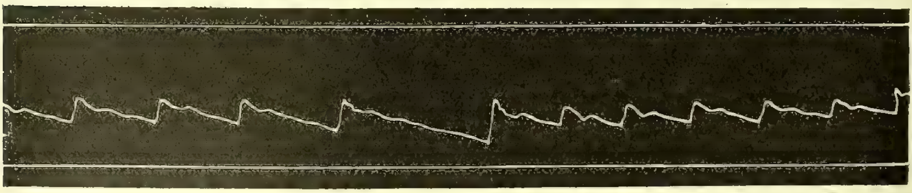
Definition.—Cardiac arrhythmia, or irregularity of the pulse, may consist in variations in the force of the successive beats or unequal duration of the intervals between the beats.

Etiology.—The causes of arrhythmia are very numerous, but all act directly or indirectly on the heart or its nervous mechanism. In certain individuals it occurs so habitually that it would seem to be an entirely normal condition in them, as it is said to be in the new-born infant. In the first place, arrhythmia may be due to diseases of the heart itself or of the vascular system. In cases of myocarditis, acute or chronic, and of myocardial degeneration, irregularity of the pulse is often the earliest and, throughout the disease, the most significant symptom. In these cases there are probably often morbid conditions of the cardiac ganglia, and in a few instances such alterations have been detected; but, on the other hand, changes of this kind do not always lead to cardiac arrhythmia. In diseases of the root of the aorta and in general arterio-sclerosis I have frequently noted marked irregularity, especially in gouty subjects. Valvular disease is a frequent cause. The pulse is more commonly irregular in disease of the mitral orifice than in aortic cases, and in the former may remain so, though compensation has been fully restored. In enlargement of the heart, arrhythmia is marked in proportion as dilatation exceeds hypertrophy.

The cardiac innervation may be disturbed, and irregularity of action result, directly or reflexly, in cerebral diseases, such as hæmorrhage, concussion, meningitis, and tumor. Very common causes of reflex disturbance are dyspepsia or other gastric disorders, especially when flatulency is present; and in diseases of the liver, the kidneys, the uterus, or ovaries arrhythmia is not infrequent.

Finally, there is the group of cases in which the functional disturbance of the heart seems independent of any morbid condition. Sometimes the irregularity of the pulse results from cardiac fatigue, as in persons who have been subjected to great and prolonged physical exertion, or in elderly individuals after ordinary strain, though in the latter myocardial or arterial diseases must always be suspected. Intemperance in the use of tea, coffee, or alcohol is a very common etiological factor, and the frequency with which excessive use of tobacco is noted in arrhythmia has led to the term "tobacco heart." Causes of this kind are especially potent in anæmic and neurasthenic persons; and in such very slight emotional excitement, as grief, terror, or anxiety, may determine a prolonged attack of palpitation and arrhythmia or irregularity alone. In a small number of cases the disorder partakes of the nature of a neurosis; more frequently it is associated with other neuroses, as exophthalmic goitre, hysteria, epilepsy, neurasthenia, and psychical affections like melancholia.

FIG. 20.

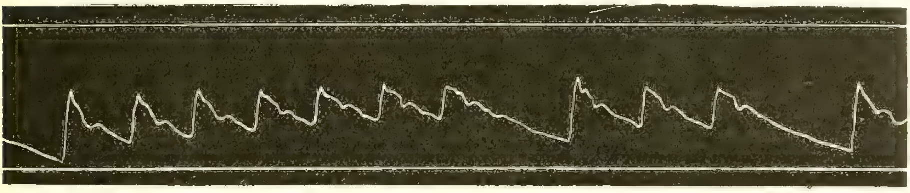


Sphygmogram from a case of General Adynamia and Incipient Phthisis, showing simple intermission of a beat.

Varieties and Symptoms.—The commonest form of irregularity is intermission of the pulse, in which an occasional beat is missed. This may be due to corresponding failure of ventricular contraction or to the weakness of the latter, in which case the pulse-rate is too feeble to be felt at the wrist. The practice of auscultation and the use of the sphygmograph in these cases is therefore important.

Sometimes the intermission occurs with great regularity, so that each second, third, or fourth beat is followed by an intermission. The term "pulsus bigeminus" is applied in cases in which every second beat is followed by an intermission. The first beat is usually stronger than the second, and the latter, as a rule, occurs before the descending stroke has reached the base line. The intermission may be due to the missing of a regular beat or to mere lengthening of the normal pause. In pulsus trigeminus every third beat is followed by an intermission. These forms are not uncommonly met with in mitral disease, but occur also in other conditions, as in the sphygmogram below from a gouty patient suffering with anterio-sclerosis, in which each third and seventh beat alternately is followed by a pause.

FIG. 21.

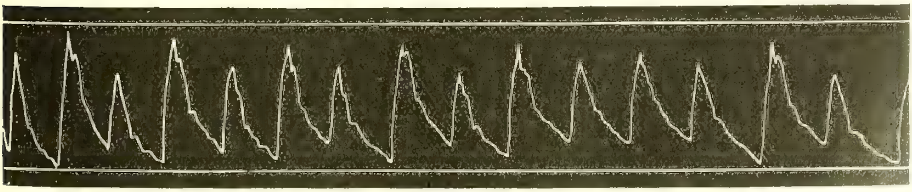


Sphygmogram showing intermission after each seventh and third beat alternately.

The cardiac rhythm is frequently disturbed by inequality in the force of successive beats. A prominent form of this is the pulsus paradoxus of Kussmaul, in which the pulse during each inspiratory effort becomes more frequent, but weaker or entirely abolished. This may result from the mechanical compression of the root of the aorta by adhesive bands of mediastino-pericarditis, but also occurs in pericarditis with effusion, in enlargement of the heart, and in certain individuals, habitually, when forced respiration is practised.

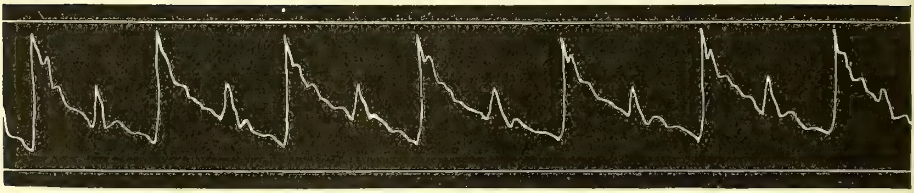
Pulsus alternans is the form in which alternate beats are weak and strong, the rhythm remaining otherwise undisturbed. The sphygmograms below were taken from cases of valvular disease.

FIG. 22.



Sphygmogram from a case of Aortic Regurgitation, with great dyspnœa, anæmia, and cardiac pain. Left ventricle considerably hypertrophied and dilated.

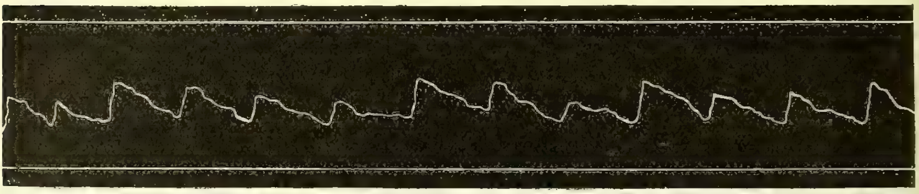
FIG. 23.



Sphygmogram from a case of Aortic and Mitral Valvular Disease, with great Hypertrophy of the Heart, showing pulsus alternans, the second beat "linked" on the downward wave of the first.

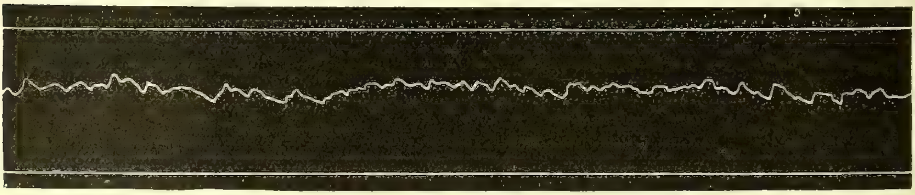
Finally, in cases of extreme dilatation, in exophthalmic goitre, in the last stages of valvular disease, and in other cases the pulse-beats become so irregular and unequal that the term delirium cordis or *folie du cœur* is highly appropriate.

FIG. 24.



Sphygmogram showing inequality in the force of successive beats. From a case of Mitral Disease with moderate disturbance of compensation.

FIG. 25.



Sphygmogram showing excessive rapidity of the pulse with extreme irregularity in force and rhythm. From a case of Aortic Valve-disease during an attack of dyspnœa.

The fœtal heart-rhythm—or, as it has been termed, embryocardia—is referred to in the description of Hypertrophy and Dilatation of the Heart. The long pause following the second is shortened, and the sounds are nearly alike, but the pulse may show no disturbance excepting the great rapidity.

Arrhythmia of the heart in itself gives rise to no symptoms, but, if marked, the underlying weakness of the heart and the failure of the circulation are manifested by the same symptoms as are seen in dilatation of the heart. Purely functional arrhythmia may exist for years without attracting the attention of the patient, but in other cases it is associated with paroxysms of palpitation or with other subjective sensations which disturb the patient's mind and lead to increase of the original trouble.

Diagnosis.—The discovery of arrhythmia in itself is attended with no difficulty when the pulse is carefully examined and auscultation practised. In certain cases, however, especially during typhoid fever, dicrotism may be so marked as to be mistaken for irregularity. The sphygmograph in these instances is of especial value.

In determining the cause of arrhythmia a thorough examination of the patient's general condition and of his habits must not be neglected. Dyspepsia, in particular, must be rigidly excluded before the irregularity is referred to organic myocardial disease. Even in cases of valvular disease coincident dyspepsia may be the occasion of increased irregularity of the pulse. Persistent arrhythmia with signs of weak heart is held by Riegel to be highly significant of chronic myocarditis.

Prognosis.—As before stated, irregularity of the pulse may be noted for years without any disturbance of the patient's health. In other cases this symptom is of ominous significance. The prognosis is to be based upon the cause of the cardiac arrhythmia. On the whole, the outlook is more grave in any form of myocardial or valvular disease in which arrhythmia occurs than when this condition is absent.

Treatment.—Rest and restricted diet are always important factors in the treatment. Sometimes a particular article of diet will be found to disagree, and must be excluded. Tea and coffee should be used with great moderation or not at all. Tobacco must be given up entirely, and the use of alcohol is to be restricted in cases in which it seems to have had a causative influence. The patient's general health may be benefited by iron, quinine, or other tonics. Nux vomica or strychnine, in particular, is valuable as a general tonic and as a nutrient to the heart-muscle. In neurotic cases bromide of potassium, valerianate of ammonium, and asafœtida exercise a happy influence. Digitalis is of value in some purely functional cases, but is especially useful in the arrhythmia and failing compensation of valvular disease and cardiac dilatation.

ANGINA PECTORIS.

SYNONYMS.—Stenocardia; Breast pang; Neuralgia of the heart.

Definition.—Angina pectoris is an affection characterized by severe pain in the region of the heart, often radiating to the left shoulder and arm, and attended with a fearful sense of impending death. It is not, as a rule, an independent disease, but occurs symptomatically in a variety of cardiac affections, though occasionally it is purely neurotic. It was first described by Heberden in 1772, and the autopsy on his first case was made by John Hunter, himself later a victim of the malady. In its pronounced form it is a rare disease, not one case having occurred in 3835 successive patients, according to Blane.

Etiology and Pathology.—The group of symptoms known as angina pectoris may be due to a number of different causes, and it is difficult to classify the latter in any wholly satisfactory manner. Two classes of cases may, however, be distinguished, both clinically and pathologically. In one the anginoid manifestations are more or less the result of organic lesions of the heart or vascular system, and the prognosis is usually of great gravity; in the other group the paroxysms are the expression of some neurosis or themselves constitute a special neurosis; and the prognosis in this case is always favorable. The first class of cases has been termed organic, symptomatic, or true angina; the second, essential, neurotic, or hysterical angina. The cases in which the painful paroxysms result from reflex action in various diseases of the internal organs have sometimes been included in the first group as symptomatic anginas, but are more rationally considered with the non-organic or neurotic variety. The cause and pathology vary widely in the two forms and will therefore be considered separately.

Organic angina is far more common than the other variety, 68 of Gauthier's 71 cases having been of this form. It is very frequently found associated with coronary sclerosis, with or without fibroid myocarditis or fatty degeneration. These are by far the commonest pathological conditions discovered at autopsy, and fibroid or fatty heart without coronary disease may give rise to anginoid paroxysms. Reference has already been made to this condition in aortic regurgitation, and more rarely other forms of valvular disease may occasion true angina. Dilatation or aneurism of the root of the aorta and atheromatous disease of the aorta are frequent causes, and particularly have I been struck by the frequency of milder forms of angina in cases of slow aortic sclerosis. Obliteration of the pericardial sac by adhesion of the layers is sometimes noted, and was the only morbid condition present in one of the severest cases I have seen. Mediastinal growths and inflammatory effusions in the mediastinum are rare causes.

Organic angina is distinctly more common in the male sex after the age of fifty than in females or in young persons of either sex. Of the cases collected by Gauthier, 126 were males and but 34 females, and of the 75 tabulated by Schütz, 64 were in persons beyond fifty years of age. The youngest recorded case is that of Dusch, which occurred in a boy of eleven years, and in which obliteration of the pericardium and a calcareous ring in the groove between the auricles and ventricles were discovered post-mortem. The preponderance of cases in males is doubtless due to the fact that their habits of life and work predispose them strongly to arterio-sclerosis and myocardial disease; and the greater frequency of the affection in old persons is due to the fact that these conditions are more common in them than in the young. Habitual indulgence in alcohol or rich foods and syphilis are powerful predisposing factors. The abuse of tobacco is also a potent cause, but acts rather upon the nervous system than by inducing degenerative cardio-vascular lesions.

Neurotic angina is met with more commonly in women, though the predisposition is not nearly so great as is that of males to the organic form. It may occur at any age, even young children being affected. It is associated with a number of neuroses, among which hysteria, hypochondriasis, epilepsy, and certain psychoses are prominent. I have seen violent cases caused by unduly frequent and close child-births; and in the male excessive fatigue, as by severe mountain climbing, has brought on severe and obstinate angina. It is occasionally observed in locomotor ataxia, and may be induced by serious emotional disturbances.

Laennec referred to groups of cases occurring together, and Kleefeld and Gelineau reported interesting epidemics which were manifestly of hysterical nature.

Intemperance in the use of alcohol and excessive smoking predispose by their unfavorable influence on the nervous system. Exposure to cold is an important factor in the determination of attacks of the form of angina described by Nothnagel under the title angina pectoris vasomotoria, to which reference will be made below.

Reflex anginoid attacks sometimes occur in hepatic, renal, uterine, and ovarian diseases, and in affections of the stomach and bowels.

The physiological cause or explanation of anginoid paroxysms probably differs in different cases, and there is doubtless some truth in each of the several theories offered: (1) Changes in the cardiac nerves have been noted by a number of observers, and angina is sometimes regarded as a neuralgia of the cardiac plexus. The close relations of the latter to the root of the aorta, and of its continuation, the coronary plexus, to the coronary arteries, seem to offer an explanation of the frequency of angina pectoris in diseases of the aorta and coronary vessels. Lancereaux, Hadden, Leroux, and Rokitansky demonstrated pathological lesions of the plexus, the vagus, and phrenic nerve, and Putjakin found alterations in the intracardiac ganglia. According to certain observers, it is possible to distinguish clinically between cases in which the vagus is involved and others in which branches of the sympathetic are implicated. (2) Spasm or cramp of the cardiac muscle naturally suggested itself to Heberden and the older observers generally, but convincing proof of the existence of such a condition is lacking. (3) Increased arterial and intracardiac tension seems undoubtedly the occasion of paroxysms in certain cases, as in aortic regurgitation and in the vasomotor angina of Nothnagel. This explanation would likewise apply to the cases of angina occurring in association with sclerosis of the aorta.

Symptomatology.—Angina pectoris is essentially a paroxysmal disease, the patient in the intervals enjoying complete health or manifesting the symptoms of the cardiac or other disease with which the angina is associated. The paroxysms come on with great suddenness, and may seize the patient without a moment's warning, or may in other instances follow after certain premonitory symptoms, such as nausea, ringing in the ears, spots before the eyes, chilliness, or paræsthesia and change of color of the skin. There is rarely an outcry at the onset, but the patient immediately suffers the most extreme pain in the lower sternal region, which he describes as burning, stabbing, or lacerating in nature. From the præcordia the pain radiates to the left shoulder and down the inner and posterior aspects of the arm to the elbow, or even to the fingers. In other cases the pain radiates to both arms or very rarely to the right alone, and sometimes it extends to the back, the neck, and the occiput. Associated with this pain, and even surpassing it in urgency, is the fearful sense of impending death which manifests itself to the observer in the features of the patient as a more or less distinctive facies. The face is ashy-gray or slightly livid, the features painfully drawn, distorted, and bedewed with cold sweat. During the paroxysm the heart's action is generally greatly excited and irregular, and the arterial tension increased. The apex-beat is forcible and diffuse, and on auscultation the peculiar *cliquetis* or *tintement métallique* may be heard beside the first sound. In other cases the heart is but little disturbed, and sometimes, as Traube observed in himself, the pulse-rate falls during the attack. The respirations are shallow, jerky, and irregular. The sense of dyspnœa may be present, but is often entirely wanting, and the respirations may be normal throughout. Abnormal sensations are occasionally experienced in the area of distribution of the pain. Numbness, formications, or other paræsthetic sensations are not uncommon, and sometimes precede or follow the attack. In the vasomotor form described by Nothnagel pallor, coldness, stiffness, or lividity are observed, and may introduce the paroxysm.

The position assumed by the patient differs in the organic and in the emotional or neurotic type of angina. In the former at the moment of onset the patient may rush to an open window or to some other point of support, or clasps his hands firmly to the præcordia, and remains fixed and quiet during the attack. In the nervous form, on the other hand, restlessness, moaning, or noisy complaint are more frequently observed.

The paroxysms may come on during sleep or during the day, and not rarely are determined by excitement, exertion, dietetic indiscretions, or, in the case of the vasomotor form, by exposure to cold. The duration of the attacks may be but a few seconds or an hour or more, and sometimes they are repeated every few minutes during several days. In other cases the intervals between attacks are weeks or months in duration.

The termination of the paroxysm is generally abrupt, like the onset, and may be marked by vomiting, discharge of flatus or copious light-colored urine, by a movement of the bowels, or by a paroxysm of coughing and expectoration. In the course of the paroxysm there may likewise be reflex disturbances of the stomach or bowels and of the voice. The patient may pass into syncope or into coma. Epileptiform seizures have occasionally been observed, and paresis of certain muscles, particularly those in the painful areas, or of one side of the body, may be present during and for some time after the attack. In the hysterical or neurotic form attacks of sciatica, hemicrania, or gastralgia may alternate with paroxysms of angina or may be associated. Occasionally the anginoid seizure is unattended by pain, and merely consists of a sense of vague fear and constriction, or the fear of death and the abnormal sensations in the arm may completely overshadow pain, though the latter be present.

Sudden death during a paroxysm has not rarely been observed, and may occur in the first attack, as in the case of the celebrated Dr. Arnold of Rugby. In these cases the heart becomes suddenly paralyzed or death may occur from rupture of the organ.

Diagnosis.—The diagnosis of angina can never be difficult. Occasionally gastralgia or a crisis of locomotor ataxia causes severe pain in the region of the heart, but the characteristic distribution of the pain and the sense of impending death are absent in these cases, and in the latter the associated symptoms would indicate the existence of locomotor ataxia.

The distinctions of the organic form or true angina from the hysterical or pseudo-angina have been especially studied by Huchard.

The important points for diagnosis are the following: The organic form occurs more frequently in the male sex and after the age of fifty years. The paroxysms are usually brought on by exertion or like cause, are rarely periodic or nocturnal; they are unaccompanied by marked nervous symptoms, but are attended by severe pain and fear of death. The attack is short, the patient remains quiet and motionless. The hysterical form may occur at any age, and is more frequent in women than in men. The attacks are spontaneous, often nocturnal and periodic, of long duration, accompanied by various nervous symptoms, but unattended with the severe pain and distress of true angina. There is instead a sense of distention of the chest. The patient is agitated and restless.

Prognosis.—The prognosis of angina pectoris depends entirely upon the cause. In the neurotic form the outlook is always favorable, and a cure is not rarely obtained. In the organic form the prognosis is extremely unfavorable, and sudden death may occur at any time during an attack.

Treatment.—Prophylaxis is always of importance to prevent the paroxysms. The patient should live as quietly and as free from all mental and physical excitement as may be possible. In cases where there is reason to suspect that the over-exertion has induced the attacks, or where neurasthenia exists, the strict rest-cure may be insisted on. The diet should be simple. Tea and coffee should, as a rule, be forbidden, though in certain cases little harm results from their moderate use. The use of tobacco must be interdicted rigidly, as also that of alcohol when the patient has suffered from intemperance in this respect. In certain cases, however, especially in feeble old men, a little wine at dinner aids digestion and acts as a most beneficial stimulant and tonic. Hard-and-fast rules cannot, therefore, be laid down, but the regulations in each case are to be governed by the condition of the general health and by the previous habits of the patient. Sometimes the patient's experience will indicate that certain articles of diet are specially and peculiarly injurious: these must of course be forbidden.

The treatment of the paroxysm must always be prompt and careful, as sudden death has often been observed. The patient should be placed in a cool and somewhat darkened room, where he will be entirely free from noise or disturbances of any kind. An ice-bag placed upon the præcordial region often gives immediate relief, and may be supplemented by swallowing of small pieces of ice. In other cases warm applications are better borne and exercise a more powerful influence on the pain. Hot cloths or hot fomentations may suffice, or where the cardiac power is greatly lessened a mustard poultice is a valuable stimulant.

The most powerful internal remedy for the control of the paroxysms is nitrite of amyl, of which three or five drops may be inhaled from a handkerchief. When appropriate, this drug acts with great rapidity, and at once reduces the pain and the fearful sense of constriction and impending death. All cases, however, are not benefited by amyl nitrite. In general, those in which arterial tension is high and in which there are evidences of vasomotor spasm are most happily influenced, but it is difficult to determine whether the drug is suitable or not until trial has been made. The administration should be discontinued immediately if the patient complains of uncomfortable fulness in the head and if the face becomes deeply congested. Patients who suffer sudden paroxysms should carry with them small beads or *perles* containing three to five drops of the remedy, which may be broken in the handkerchief and immediately inhaled when the attack begins.

Nitro-glycerin may also be used during the paroxysm, and where rapid action is desired, as is generally the case, the remedy may be given hypodermically. One or two drops of the officinal 1 per cent. solution may be given at a dose, and may be increased when the patient has become accustomed to its use. Generally, however, nitrite of amyl is more prompt and more powerful, and is therefore preferable during the paroxysm. In the intervals nitro-glycerin is of great value in cases in which the arterial tension is habitually high. Nitrite of sodium is also a valuable remedy, and may be given in doses of from one to three grains.

In cases in which the nitrites fail to control the paroxysm morphine may be used hypodermically, and it is usually well to add a small dose of atropine (gr. $\frac{1}{200}$ to gr. $\frac{1}{150}$). In few cases does this fail to relieve the pain and check the paroxysm, but it is always advisable to try the nitrites first.

When the heart's action is decidedly weak, diffusible stimulants, such as ammonia, Hoffmann's anodyne, ether, and alcohol, with hot applications to the præcordia, are generally useful. In the vasomotor form Nothnagel recommends friction of the extremities, stimulating liniments, and hot foot or general baths.

Injections of cocaine hydrochlorate (gr. $\frac{1}{3}$ to gr. $\frac{1}{2}$) have been recommended, but have not proved of great value. Inhalations of chloroform and ether have also been recommended, but are distinctly dangerous, and should not be used.

During the interval between attacks nitro-glycerin or nitrite of sodium may be used continuously when the arterial tension is persistently high. The iodide of potassium is perhaps the most valuable remedy in cases in which arteriosclerosis is present, and is especially to be used when there is a history of syphilis. The drug may be used in doses of five or ten grains three times daily, with occasional intermissions to prevent toxic symptoms. Preparations of mercury may also be useful in syphilitic cases, but are far more uncertain than the iodide. In gouty cases, besides the strict regulation of the diet, alkalies or natural mineral waters are often of service. Digitalis is, as a rule, to be avoided on account of its tendency to increase arterial tension, but may be useful in cases, such as aortic regurgitation, in which the heart-power is failing and intracardial pressure is increased. Small doses of the tincture (♏. iii to ♏. v), with equal quantities of tincture of nux vomica, will usually suffice.

In cases of pseudo-angina the treatment is to be directed to the general nervous condition of the patient, and the sedative nervines, like bromide of potash, valerian, or valerianate of ammonium, and asafœtida, are especially useful. At the same time, the general condition of the patient is benefited by tonics. I have obtained particularly happy results from the chloride of gold and sodium, which may be given hypodermically in doses of a twentieth to a fifth of a grain. The drug. however, sometimes proves irritating, and may then be given by the mouth. Iron and arsenic are useful when anæmia is marked.

Electricity in some cases proves valuable. The descending galvanic current, the positive pole being placed over the neck, the negative over the region of the heart, seems especially useful. Static electricity is sometimes employed. Lastly, I have derived decided benefit in a number of cases from repeated applications of the thermo-cautery, at suitable intervals, over the præcordia and the aortic area.

Exophthalmic Goitre.

Synonyms.—Graves's disease; Basedow's disease: Tachycardia strumosa exophthalmica.

Definition and History.—Exophthalmic goitre is an affection characterized by enlargement of the thyroid gland, protrusion of the eyeballs, and by various nervous manifestations, including tachycardia. It is essentially a nervous disease, which for the present is to be regarded as a neurosis affecting particularly the cardiac innervation. The disease was first completely described by Graves of Dublin in 1835, and independently by Basedow in Germany in 1840.

Etiology.—No age, excepting infancy, is exempt from the disease, but it is most common between fifteen and fifty years. The average age in Augustus Eshner's table of 227 cases was between thirty and thirty-one years. In children the symptoms are apt not to be typical, though some years since I reported a fully-developed case in a girl ten years old. Women are far more prone to be affected than men.

Direct heredity plays but a small part as an etiological factor, though occasionally several members of one family have been affected. Indirect heredity through family tendency to nervous disease is more important. Anæmia, exhausting discharges, fright, and mental depression may all act as predisposing causes, but the patient may be seized while apparently in the best of health. Prolonged mental impressions of a depressing character, as grief, anxiety, and the like, are particularly frequent.

I have been much struck with the frequency with which marked gastro-intestinal catarrh has preceded the development of this neurosis. The exhausting effects of impaired assimilation with diarrhœal discharges naturally enhance the influence of the severe irritation of the nerves of the stomach and intestines.

Morbid Anatomy.—The most important lesion discovered is the enlargement of the thyroid gland. As a rule, this is more or less uniform, affecting all portions of the organ, and on section it is found that the vascular structure of the gland is greatly in excess. The morbid anatomy, therefore, essentially consists of a teleangiectatic or cavernous condition of the blood-vessels of the thyroid gland.

Various changes in the sympathetic and central nervous systems have been found post-mortem, but none have been constant, and none explain all of the symptoms. Hale White reports a case in which there were hæmorrhages in the floor of the fourth ventricle, and Filehne succeeded in producing the disease experimentally in one case by division of the restiform bodies. While the medulla, therefore, is possibly the essential seat of the affection, it is not yet proved to be so, and no theory as yet announced satisfactorily explains the disease.

The secondary cardiac changes will be referred to in the Symptomatology.

Symptomatology.—The cardinal symptoms are rapid heart, exophthalmos, and enlargement of the thyroid gland. The onset may be acute or gradual. In a case of J. H. Lloyd's at the Philadelphia Hospital, in whom for some time there had been slight prominence of the eyeballs, there suddenly developed rapid action of the heart, marked arterial throbbing, enlargement of the thyroid, vomiting, and diarrhœa, with death in three days. Usually, however, the onset is slow, and the symptoms referable to the vascular system are the first to appear.

The patient complains of paroxysms of palpitation with moderate dyspnœa. On examination the heart is found to be beating rapidly and violently, the apex-beat is in its usual situation, and sometimes there is a soft systolic murmur at the base. The pulse is rapid and often highly irregular. At first it is sudden and bounding, but when secondary changes have taken place in the heart it may become quite feeble. The carotid vessels throb visibly, and a systolic murmur is heard in them. The capillary pulse of Quincke and venous undulations may also be present. At first the rapidity of the heart's action varies greatly, and is strongly influenced by emotion and exertion, but later it is persistently rapid, sometimes reaching 200 beats per minute. As time passes, signs of cardiac hypertrophy and dilatation appear. At first the sounds become excessively loud, and in one case could be heard four feet from the patient. Later, with increasing dilatation, the sounds are less distinct, and a murmur of mitral insufficiency is not uncommonly developed. Slight mitral endocarditis is occasionally found post-mortem. The pulse in the later stages loses greatly in force, and may become excessively irregular.

The enlargement of the thyroid usually proceeds slowly, both lobes being affected, but rarely the isthmus alone. The enlargement is never so marked as in advanced cases of ordinary goitre, and the right lobe is apt to be larger than the left. The size may vary from day to day, or even within a few hours, being largest when palpitation is worst. The superficial veins are dilated, the arteries and the gland itself pulsate strongly, and there may be a distinct arterial thrill, as well as a loud, blowing, sometimes musical, murmur. Cases are occasionally met with in which the enlargement of the thyroid is absent throughout the entire course of the disease.

The exophthalmos gives the patient a peculiarly repulsive appearance. Rarely, it is the first symptom, but much more commonly it follows the cardiac disturbance. Exophthalmos is never unilateral, but may be more marked on one side than on the other. It often varies in degree with the varying cardiac conditions. A remarkable symptom in connection with the eyes is named, after its discoverer, Graefe's sign. On looking downward the upper lid does not follow the fall as in health, but remains in a state of spastic elevation. In some cases the upper lid simply lags behind the moving ball. This symptom may be present when exophthalmos is slight, but it is often absent. The fall of the lid on closing the eye is not interfered with. Sometimes the involuntary winking of the eye is lost and the palpebral angle is wider than normal from retraction of the upper lid (Stellwag's sign). Conjunctivitis, and even sloughing of the cornea, may occur, due probably to the want of proper protection. The sensibility of the cornea is apt to be diminished on account of the rapid drying of the tears on the exposed surface. The only important ophthalmoscopic appearance is pulsation of the retinal vessels. Möbius has occasionally found insufficiency of convergence.

Among the minor symptoms tremor, either general or limited to one part, ranks first. It varies much in character, sometimes greatly resembling that of paralysis agitans, but more usually it is coarse and jerky, and occurs only on movement. A subjective feeling of heat and fulness, with throbbing in the head, is very frequent, especially after slight exertion, and may be accompanied by a rise of the temperature of from one to three degrees. Violent blushing and sweating may be induced by slight effort or emotion, and both may be unilateral. The *tâche cerebrale* of Trousseau may sometimes be evoked. Recently Charcot has shown that there is decreased electrical resistance in the skin, due probably to its saturation by fluid from the dilated capillaries. Headache, tinnitus aurium, and insomnia are symptoms occasionally observed.

There is sometimes a copious secretion of pale urine before a paroxysm of palpitation. Glycosuria occurs quite frequently, and intermittent albuminuria at times. Gowers speaks of three cases in which there was enlargement of the lymphatic glands similar to that met with in lymphadenoma. Dark pigmented areas or diffuse bronzing of the skin, and indeed true Addison's disease, are sometimes present. In many cases there is mild melancholia or mental irritability, and serious melancholia or violent acute mania may occur. Graves's disease may be present in paretic dementia, and cases of recurrent insanity are reported in which with each outbreak all the symptoms of the disease under consideration have appeared. Hysterical manifestations are common, and chorea, epilepsy, migraine, and various neuralgias are occasional complications.

Of the three cardinal symptoms, the rapid action of the heart is the most constant and the most important. Indeed, so frequently is it the most prominent symptom that every case of tachycardia should be closely studied for other evidences of the disease. Some authors are even of opinion that paroxysmal tachycardia is generally if not always a masked form of Graves's disease. Sometimes rapid heart and enlargement of the thyroid alone are present to the end of the disease.

Diagnosis.—The differential diagnosis from common goitre is usually easy on account of the absence of cardiac and ocular symptoms in the latter. It must not, however, be forgotten that a large bronchocele may by pressure upon the vagus and cervical sympathetic cause exophthalmos and mydriasis on the same side on which the enlargement is greater, and also rapid action of the heart. Aneurism of the carotid artery may simulate goitre to some degree, but needs only to be remembered to avoid error.

Course and Prognosis.—The duration of the disease varies greatly. As in the case before cited, it may run its course and terminate fatally a few days after the first pronounced symptoms, but, as a rule, it lasts several years, sometimes with periods of lessening or complete intermission of the symptoms. Probably 20 or 25 per cent. of the cases recover, but even in those which do not recover there may be distinct and lasting improvement. Failure of nutrition and organic heart disease make the prognosis distinctly more grave. The mortality is greater in men than in women. Death is sometimes very sudden, and may be preceded by excessive elevation of the temperature.

Treatment.—Hygienic measures are in all cases of the utmost importance. Emotional disturbances play so important a part in the causation and increase of the disease that the patient must be most carefully guarded against any unnecessary excitement, and if possible should be removed from his customary surroundings. Change of climate often exercises a highly beneficial influence upon the disease, and doubtless largely by securing mental tranquillity. The diet should be plain but nutritious, and in bad cases advantage is sometimes derived from an absolute milk diet. The patient must be placed at rest, and in severe cases should be confined to bed. Gentle daily massage will give the muscles exercise and improve their tone without exertion on the part of the patient.

Of the remedies suggested for the treatment of this disease, digitalis commends itself at once as likely to control and steady the action of the heart, and in some cases it acts most happily. Small doses should be used, and the remedy may be continued for a long time if the action be favorable. Strophanthus and sparteine have been used as substitutes for digitalis, but are generally valueless when digitalis fails. Excessive palpitation is sometimes controlled by veratrum viride or aconite. More useful, however, than any of these drugs is belladonna, which may be administered in the form of the tincture or of the alkaloid atropine. It should be used in ascending doses and until distinct symptoms of the physiological action have appeared.

In cases in which nervous excitability is marked, and particularly in hysterical cases, bromide of potassium, valerian, and similar remedies may be employed with advantage, but are only useful as temporary expedients.

When the cardiac action is very rapid, the local application of cold to the præcordia is of great value. An ice-bag or Leiter's coils may be placed over the heart, or, better still, friction may be made with a piece of ice. A smooth piece should be briskly rubbed over the præcordia once, twice, or three times daily, the time being increased a little each day until fifteen minutes is reached.

Recently a few cases have been treated by feeding with raw thyroid glands of the sheep and by hypodermic injection of thyroid extract, but with unfavorable result. In one case of my own the subjective sensations increased so much that the treatment had to be discontinued. According to J. J. Putnam, thyroidectomy has been performed some 40 times with but two deaths and a very large percentage of cures or improvements. He holds that any means that will prevent a considerable number of excitations from reaching the irritable centres is likely to be beneficial by securing for them a partial physiological rest. Nothing so effectually accomplishes this as thyroidectomy, but the operation is attended with risk of death and a greater risk of considerable temporary prostration and laryngeal paresis.

Galvanism of the neck has been used with success in some cases, and should always be tried if belladonna, digitalis, and the local use of cold have failed to bring improvement.

MALPOSITIONS AND CONGENITAL AFFECTIONS OF THE HEART.

BY WILLIAM PEPPER.

MALPOSITIONS OF THE HEART.

MALPOSITION or displacement of the heart may be congenital or acquired in later life.

Of congenital malpositions, the most important is *dextrocardia* or *dexiocardia*, a condition in which the heart is situated to the right instead of to the left of the middle line. The apex-beat is found in the fifth interspace of the right side, and the cardiac dulness has the same boundaries on the right side as it has customarily on the left. The arch of the aorta curves over the right instead of the left bronchus, and the descending and abdominal aorta are found on the right of the spinal column. In nearly all cases the other viscera are also displaced, the liver occupying the left, the spleen the right, hypochondriac region, and the stomach and intestines showing a like inversion of their normal position. Occasionally one or other of the viscera may retain the usual position, and in rare cases the heart alone is displaced.

Mesocardia, the condition in which the heart is found in the middle line, is normal in the fœtus, and may sometimes persist after birth. In the majority of such cases congenital affections of the heart itself are present.

A more serious form of congenital misplacement is that known as *ectopia cordis*, in which the heart is found immediately beneath the surface or completely exposed in the thoracic, abdominal, or cervical regions. In thoracic ectopia the sternum is divided in the middle line, and the heart is found beating beneath the skin, or the organ may be completely exposed to view with but a partial covering of pericardium. There may, in extreme cases, be even complete absence of pericardial covering. The lesser grades, in which the heart is covered by the skin, are not incompatible with prolonged life, but in cases of full exposure the child lives but a few hours.

In cases of ventral or abdominal ectopia the heart may occupy a position anterior to the stomach, and has occasionally been found as one of the constituents of an umbilical hernia. Peacock reported a case of abdominal malposition of the heart in a man aged forty-seven years, and Rezek one in a man of thirty-two years.

The rarest and most serious malposition is that in which the heart occupies the cervical region. In certain animals this condition may exist without influencing the general health, as I have observed in a cow which has been kept for several years past at the Veterinary Department of the University of Pennsylvania.

Acquired displacements of the heart result from diseases of the neighboring structures.

The position of the heart is elevated in a variety of conditions which cause distention of the abdomen and elevation of the arch of the diaphragm, such as tympany, ascites, abdominal tumors, or enlargement of the liver or spleen. I have seen the heart pushed up as far as the second interspace in a case of extreme tympany in typhoid fever, and there is now in my wards at the University Hospital a boy whose heart occupies a similar position as a result of ascites with tympany. When the displacement occurs suddenly, the action of the heart may be seriously disturbed, whereas in cases of slow enlargement of the abdomen, as in the second case referred to, the heart adapts itself to the changed condition and little disturbance of its action may occur. Upward displacement may also result from mediastinal growths or from contraction of the apex of the lungs.

Downward displacement occurs when the diaphragm is depressed by emphysema of the lungs, and when the heart is pushed downward by the weight of an aortic aneurism. A degree of lowering may also be observed in emaciated persons in whom the various abdominal organs, as well as the diaphragm and heart, sink downward. The action of the heart is rarely disturbed in these cases.

Displacement to the right or left may result from the pressure of air or effusions in the pleural cavities. Tumors or enlargements of the spleen or left lobe of the liver may also affect the position of the heart. Lateral displacement may also occur in cases of contraction or cirrhosis of one of the lungs with pleural adhesions. In the latter instances the displacement results from traction or from compensatory enlargement of the opposite lung. In left-sided pleurisy with effusion it is not unusual to find the heart as far to the right as the mammary line, and it may be considerably rotated on its axis. In acute cases there is usually more or less disturbance of the heart's action, but in cirrhosis of the lung or in cases of pleural adhesions marked displacement may give rise to few if any symptoms.

The heart is sometimes pushed forward against the chest-wall by a growth in the posterior mediastinum, and may by its vigorous impulse and increased area of dulness simulate hypertrophy. The character of the sounds and the examination of the pulse will, however, often lead to a correct interpretation of the condition present.

Backward displacement may result from new-growths or abscess of the anterior mediastinum and from pericardial effusion.

CONGENITAL AFFECTIONS OF THE HEART.

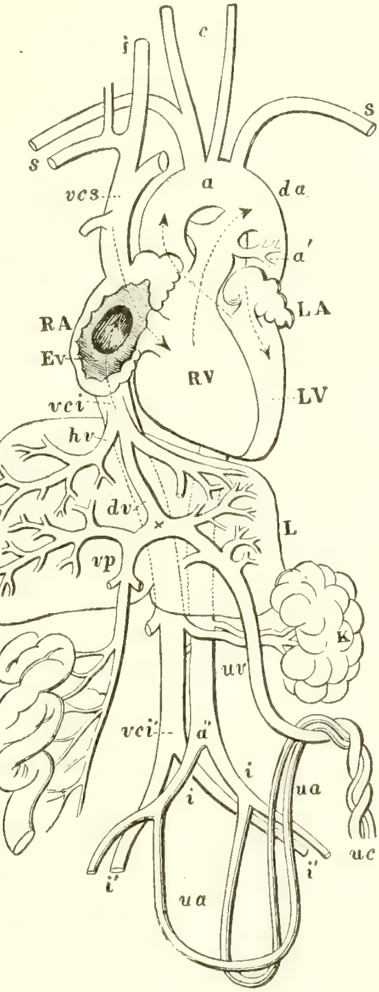
SYNONYMS.—Congenital cyanosis; Morbus cœruleus; Blue disease. These names are, strictly speaking, applicable only to those cases of congenital cardiac affections in which cyanosis is a symptom, but, as this occurs in a great majority of all instances, the names may be taken as synonymous with the one used as the title of this article.

Definition.—Congenital affections of the heart are of two kinds—those which result from fœtal endocarditis and myocarditis, and which, for the most part, affect the valves and orifices; and those which are strictly developmental defects, and which may affect any part of the organ. Frequently there is a combination of endocarditis with defective development, and then the difficulty of determining the proportion of each element in the case is very great, but even where the lesion is a simple one it is often difficult to say whether it is inflammatory or not.

Etiology.—The causes which lead to endocardial or myocardial inflammation in the fœtus are entirely obscure. Undoubtedly the condition of the mother, and especially a depraved state of the blood, may play a part, but the exact importance of such influences or the nature of the pathological conditions cannot be determined. Disturbance of the fœtal circulation is likewise a factor in certain cases, as is evident from the great proneness of malformed valves to take on secondary inflammatory changes. The unusual strain to which the affected parts have been subjected in cases of fœtal endocarditis is comparable to the increased pressure and strain which are operative in endocarditis and valvular disease resulting from excessive physical exertion in late life.

Malformations of the heart result from retardation or perversion of the normal evolutional changes, and are often easily accounted for when the development of the heart is recalled. It may be remembered that in the early stages of fœtal life the heart consists of a tube bent upon itself and roughly simulating an S-shape. Later, a transverse constriction divides it into an auricular and a ventricular cavity, each of which is subsequently divided into two by longitudinal septa. The aorta and pulmonary artery are formed by a similar partitioning of the common truncus arteriosus. In the later months and for a brief period after birth the adult condition of the heart is present, with the exception that there is an opening through the auricular septum, the foramen ovale, and a communication between the aorta and pulmonary artery, the ductus arteriosus Botalli. Finally, these are obliterated soon after birth, and the development of the heart is completed. A knowledge of these points will serve to elucidate some of the defects described below. The foramen ovale and ductus Botalli are necessary during fœtal life, because the pulmonary circulation has not been established, and the blood must therefore be diverted from the lungs. (See Fig. 10.) If for any reason—as, for example, in pulmonary collapse, pneumonia, or pulmonary stenosis—the circulation through the pulmonary artery remains impeded at birth, the fœtal condition of the heart persists, and may become permanent. In these cases the causes of the congenital cardiac affection are therefore clear; but of the causes leading to strictly imperfect development or malformation we know but little. Heredity plays an important part, and in certain families a number of cases may occur in succession. Male children are more often affected than females. Lesions of the right heart are far more common than those of the left side, the reason being that during fœtal life the former is the part of the organ which is functionally more active, and upon which any strain will fall with greatest force.

Fig. 26.



Diagrammatic View of Fœtal Circulation: RA, right auricle of the heart; RV, right ventricle; LA, left auricle; Ev, Eustachian valve; LV, left ventricle; L, liver; K, left kidney; I, portion of small intestine; *a*, arch of the aorta; *a'*, its dorsal part; *a''*, lower end; *vcs*, superior vena cava; *vci*, inferior vena where it joins the right auricle; *vci'*, its lower end; *s*, subclavian vessels; *j*, right jugular vein; *c*, common carotid arteries; four curved dotted arrow lines are carried through the aortic and pulmonary opening, and the auriculo-ventricular orifices; *da*, opposite to the one passing through the pulmonary artery, marks the place of the ductus arteriosus; a similar arrow line is shown passing from the vena cava inferior through the fossa ovalis of the right auricle, and the foramen ovale into the left auricle; *hv*, the hepatic veins; *vp*, vena portæ; *x* to *vci*, the ductus venosus; *uv*, the umbilical vein; *ua*, umbilical arteries; *uc*, umbilical cord cut short; *i*, *i'*, iliac vessels.

Morbid Anatomy.—The anatomical changes in a given case are generally more or less complicated; only occasionally is a single lesion observed. Moreover, secondary changes are frequently induced which obscure the primary condition.

The developmental abnormalities for the most part consist in some defective condition of the interventricular or interauricular septum or in faulty division of the primary truncus arteriosus. Other errors in development are occasionally observed, but are of minor importance.

Fœtal endocarditis is of the indurative or sclerotic type in a great majority of cases, though instances have been observed in which vegetations of acute endocarditis were detected in the new-born child. Such cases as these, however, are rare. Generally the endocardium of the affected part is thickened and contracted, and secondary degenerative changes, fatty or calcareous in nature, are sometimes observed. These processes may occur in previously normal valves, but are especially apt to arise in those presenting some malformation; and the relative importance assignable to inflammation or to errors of structure is therefore difficult to determine. Inflammatory changes are also frequent about the edges of perforations of the septa, which become thickened and roughened or present the appearances of acute verrucose endocarditis. The myocardium beneath the affected portions likewise suffers hyperplastic changes, and by its subsequent contraction may contribute to increase of the cardiac defect. Occasionally myocarditis arises primarily and unassociated with endocarditis or imperfections in development.

It will be necessary now to consider the more important of the congenital affections separately, though in reality several are usually found combined.

1. Stenosis and atresia of the pulmonary orifice, the pulmonary artery, and the right conus arteriosus are the most common and important of the affections under consideration. Of 181 cases of congenital cardiac affections, Peacock found one or other of the above conditions in 119.

Stenosis of the pulmonary orifice is generally the result of endocarditis. The valve-segments may be greatly thickened and sclerotic, or may be united at their edges so as to form a diaphragm with a narrow orifice. Secondary degenerative conditions and calcification are not rarely observed. The conus arteriosus may be narrowed by a contracting zone of myocarditis and endocarditis, but more frequently these are secondary changes, the original stenosis being due to improper division of the truncus arteriosus. Sometimes the pulmonary artery itself is stenotic, or in extreme cases its lumen may be completely obliterated, a fibrous cord alone remaining. These changes may affect a considerable portion of the vessel, and even one of its branches may be contracted or obliterated.

Complete atresia of the orifice, the conus, or the artery is much less common than is stenosis.

A moderate degree of stenosis may exist without any other defect, but in a great majority of cases perforation of the ventricular septum or a patulous foramen ovale coexists. When the pulmonary stenosis is developed early in fœtal life, the ventricular septum, as well as the foramen ovale and ductus Botalli, remains open ; but if the ventricular septum has already been closed, the latter two continue patulous, and the pulmonary circulation is maintained through the left heart, the aorta, and the ductus Botalli. The defect in the ventricular septum is generally, as Rokitansky showed, in its anterior portion, and the septum may at the same time be so deviated to the left that the aorta arises practically from both ventricles. Occasionally the septum is almost completely absent. The communication between the auricles is usually through the foramen ovale, but there may be defects in the septum proper.

Stenosis about the pulmonary orifice occasions hypertrophy of the right ventricle, which may compensate the defect for many years. If, however, there be complete atresia and an imperforate septum, the ventricle becomes atrophied from disuse.

2. Defects of the auricular septum are always due to some error or arrest of development, though the edges of the perforation are often greatly changed by secondary endocardial and myocardial inflammation. Normally, the membrane of the foramen ovale becomes attached, and closes the orifice soon after birth, when the pressure in the two auricles becomes equalized by the establishment of the lesser circulation. If for any reason such equalization of the pressure is prevented, the foramen remains partly or wholly patulous, as in the case of pulmonary, tricuspid, mitral, or aortic stenosis, or in pneumonia or pulmonary atelectasis of the new-born child. A patulous condition of the foramen ovale is therefore usually a secondary lesion, but it is sometimes detected in persons who have lived to full adult or advanced age, and in whom no other lesion is discovered. The perforation may consists of a narrow, slit-like aperture where the membrane is unattached, through which a probe is passed with difficulty, or the membrane may be wholly wanting, when the point of a finger may sometimes be passed from one auricle into the other. Occasionally the membrane is penetrated by numerous slit-like perforations. The septum proper is defective or completely absent in rare cases, and there may thus be but a single auricular chamber.

Premature closure of the foramen ovale was noted by Vieussens, and has been observed in a few cases since his time.

3. A defective ventricular septum is a very common condition, which may exist alone, though more frequently associated with pulmonary stenosis, perforate foramen ovale, and patulous ductus Botalli. The usual position of the perforation is in the anterior portion of the septum ; that is, in front of the membranous portion, or, as it has been termed, the "undefended space." There is thus established a communication between the two ventricles, or in rare instances between one ventricle and the opposite auricle, or the four cavities may communicate with each other through a common opening. A few observers have described cases in which there were abnormal communications between the two ventricles by long and tortuous sinuses. Sometimes there are several perforations, and occasionally the septum may be completely absent. The edges about the perforation may be thickened and inflamed or rarely

ulcerated, so that there may be difficulty in deciding whether the communication is inflammatory or not.

4. Congenital lesions at the tricuspid orifice are much less common than was formerly believed. The investigations of Fenwick and Leudet, among others, were important in showing that tricuspid stenosis frequently arises in youth or early adult age. Congenital lesions at the tricuspid orifice are usually combined with other defects. In some cases they seem entirely due to fœtal endocarditis; in others they are purely errors in development. In cases of stenosis the valve and the tissue about the orifice are greatly thickened and sclerotic, and secondary changes, such as fatty degeneration and calcification, may be observed. Sometimes the cusps are united, so that the orifice is closed by a diaphragm, with but a narrow perforation. Congenital tricuspid regurgitation is extremely rare, but three cases having been recorded (Eichhorst). In one of these (Ebstein's) there was almost complete absence of the valve, only small stumps indicating its usual situation.

Associated with tricuspid stenosis there is almost always perforation of the auricular septum, and when the stenosis is extreme the pulmonary circulation is made possible by accompanying defects in the ventricular septum or by a patulous ductus Botalli and foramen ovale. The latter is invariably present when there is pulmonary stenosis in addition to the tricuspid lesion.

The right ventricle becomes more or less atrophied, especially when the pulmonary orifice is also stenotic.

5. Persistency of the ductus arteriosus Botalli is a very common affection in association with other lesions, but is rarely present alone. Occasionally, however, it is wholly unassociated with other defects, and may be found postmortem in persons of advanced age when no cardiac lesion had been suspected. In a great majority of cases the foramen ovale is patulous or the pulmonary orifice constricted, and very frequently there are defects in the ventricular septum. The duct is sometimes abnormally long, at other times greatly reduced in length, and generally more or less dilated, especially at the aortic end, where it may present a funnel-like enlargement. The duct may be so short or contracted that the aorta communicates directly with the pulmonary artery through a foramen; and in a few instances direct communications between these vessels have been observed at other points. Absence of the ductus Botalli is a rare condition, as is also premature obliteration of its lumen. The latter condition is sometimes observed in cases of pulmonary stenosis, when the supply of blood to the pulmonary artery is deficient.

6. Congenital narrowing at the aortic orifice is a rare condition. Rauchfuss was able to collect thirty-three cases, and found that in the majority of those in which the narrowing occurred at the orifice there were evidences of inflammatory contraction. On the other hand, according to Dilg, imperfect division of the truncus arteriosus occasioned narrowing of the conus arteriosus in 8 of 15 cases, the other 7 probably resulting from endocarditis and myocarditis. In cases of inflammatory narrowing the septum ventriculorum is generally closed, the foramen ovale, however, remaining patulous; but when improper division

of the truncus arteriosus causes the defect, the ventricular septum is usually also imperfect, and in these cases atresia is more common than stenosis. The ductus arteriosus is almost necessarily open in cases of considerable stenosis or atresia. The mitral orifice may be narrowed, and the valve segments and the surrounding tissues thickened at the same time.

The left ventricle is usually smaller than normal, especially when mitral stenosis is present, and the right ventricle is hypertrophied.

7. Narrowing at the mitral orifice is a rare condition. Usually it is due to fœtal endocarditis, and often regurgitation is associated. The septum of the ventricles is generally defective and the foramen ovale patulous.

8. Transposition of the vessels springing from the heart is very rarely observed. The aorta may arise from the right ventricle, and the pulmonary artery from the left, or the venous trunks may be similarly transposed. In either case the foramen ovale must remain patulous, and generally perforations are present in the ventricular septum. In case of complete transposition of veins and arteries, the right heart assumes the function of the left, and *vice versa*, no other malformation being required to equalize the circulation.

Certain anomalous conditions may be found about the valves, which in themselves are of little importance, but which predispose to subsequent endocarditis. The most important of these is increase or diminution in the number of segments. Not uncommonly four leaflets are seen instead of three in the semilunar valves, especially the pulmonary, and in a few instances as many as five segments have been observed ; on the other hand, there may be but two. In these cases the function of the valve may be perfectly performed, but very frequently the leaflets undergo secondary inflammatory changes and become obstructive or incompetent. The complete absence of a valve has sometimes been discovered, and more commonly the point of insertion of the leaflets is somewhat abnormal.

Symptomatology.—The most important symptom, and the one from which the disease has received its clinical designation, is cyanosis, which occurs in a great majority of cases. Considerable dispute has arisen regarding the cause of this symptom, but an impartial review of the evidence is conclusive that both the venous condition of the blood and the overfilling of the venous system are important factors.

Cyanosis usually appears within a few days of birth, and may be noted when the child is born. Sometimes, however, there is so little duskiness of the skin that it may be overlooked, excepting when the child cries, and in some patients there may never be any blueness for many years. Usually, however, in marked cases, cyanosis becomes decided, and the skin may be intensely dark. The cyanotic condition of the skin may, however, appear and disappear with varying conditions of the child's general health. Bronchitis and pulmonary affections are especially liable to increase the cyanosis, whereas anæmia may cause a complete disappearance. If the child lives a few years, the features soon show decided characteristics of stasis of the circulation. The nose and lips are usually thick ; the complexion, if not cyanotic, is constantly clouded or dusky ; the finger-ends are swollen or " clubbed," and the nails sharply curved. The development of the entire body is retarded, and the child remains small, with flabby muscles and little fat. Even the mental development is rarely complete. Occasionally there is protrusion of the eyes, or exophthalmos.

The surface temperature is often reduced below the normal and the exposure to damp and cold is badly borne. The pulse is usually small and weak, especially upon the left side. Increasing cardiac weakness is manifested by dyspnœa, by still greater weakness of the pulse, and by deepening cyanosis, with paroxysms of apparent asphyxia or asthmatic seizures. Dropsy very rarely occurs. Occasionally convulsions supervene and frequently lead to a rapidly fatal termination. Hæmorrhages may occur from the nose, the gums, or lungs, and may afford a certain degree of temporary relief to the cardiac embarrassment. Cerebral hæmorrhage may occur during convulsions or quite apart from these.

Very frequently there is constant cough, and pulmonary diseases are prone to supervene.

Diagnosis and Physical Signs.—The diagnosis of congenital affection of the heart is usually determined by the presence of cyanosis, but it must be recalled that an imperfect septum or a patulous foramen ovale may exist to the end of the patient's life without the least cyanosis. On the other hand, imperfect expansion of the lungs without any cardiac affection, may cause a slight degree of blueness of the skin, which may persist for many days. The diagnosis must therefore in every case be confirmed by physical examination of the heart. The distinction of the different lesions is sometimes possible, but in the great majority of cases can only be approximately accurate, on account of the frequency of combined lesions, and on account of the great difficulty in properly locating and timing the murmurs in young infants. In older patients more satisfactory results are obtained. If the child has passed the twelfth year, the lesion in a large proportion of the cases will be found to be stenosis at the pulmonary orifice.

Stenosis at the pulmonary orifice leads to hypertrophy of the right ventricle, with bulging of the lower sternal region and dulness extending beyond the right border of the sternum. There is usually more or less pulsation to the right of the apex, and sometimes in the epigastric space. A thrill may be detected in the second intercostal space of the left side, and in the same situation there is usually heard a systolic murmur, which may be transmitted to the vessels of the neck, especially those of the left side. The second sound of the heart may be completely absent or greatly weakened at the root of the pulmonary artery. Cyanosis beginning soon after birth, with enlargement of the right ventricle, a systolic murmur plainly heard in the pulmonary region, and a feeble second sound, indicate with some degree of probability the existence of pulmonary stenosis. If the child be above twelve years, the probability becomes extremely great.

In cases of patulous foramen ovale there may be no abnormal signs whatever, or the signs of coexisting valvular affections may completely obscure those of the defect in the septum. A presystolic or a systolic murmur may be heard to the left of the sternum in the third interspace, and posteriorly over the vertebræ, but the time of the murmur is difficult to determine. When cyanosis without any abnormal physical sign is present, perforation of the septa must always be suspected.

When the ventricular septum is imperfect, there may be a loud systolic murmur, heard with maximum intensity near the apex, and transmitted to the axilla and the back. The first sound of the heart may be completely obscured.

The signs observed in cases of patulous ductus Botalli are often largely the result of associated conditions. The right ventricle is usually enlarged, and there may be dilatation of the root of the pulmonary artery, causing increase of dulness to the left of the sternum and a palpable thrill. A systolic, or occasionally a diastolic, murmur is heard over the upper part of the sternum, and the second pulmonic sound is accentuated.

Congenital aortic and mitral lesions are so rarely met with that their diagnosis scarcely merits consideration. In cases in which there are combined lesions the physical signs may be so varied and complicated that it is impossible to arrive at any determination of the lesions present.

Prognosis.—The prognosis of congenital cardiac disease is extremely grave: most cases terminate within a few days of birth, and of the more favorable few survive the first decade of life. Even when life is protracted beyond this point, the condition of the patient is apt to grow increasingly distressing. Occasionally a defective auricular or ventricular septum, or a patulous ductus Botalli is found post-mortem in persons of advanced age in whom no cardiac lesion was suspected, but such instances are rare. Of the common forms, the prognosis is best in pulmonary stenosis. Slight degrees of stenosis at this orifice or at the aortic orifice are not incompatible with long life.

A majority of the cases which do not die soon after birth develop phthisis or succumb to some other pulmonary affection. Cerebral hæmorrhage and hæmoptysis are among the occasional causes of death. Not rarely convulsions immediately precede the fatal termination.

Treatment.—Hygienic measures form the most important element in treatment. The child must be prevented from making unnecessary exertions, and must be protected against mental disturbances of all kinds. The diet should be simple and nutritious, and care is to be taken that attacks of bronchitis may be avoided. The clothing next the skin should be woollen and carefully adapted in its weight to the season of the year. Exposure to drafts and chilling are always badly borne, and must therefore be avoided. Constipation may be induced by the sedentary life required, and may be obviated by the use of mild salines and by regulation of the diet.

Tonics will usually be necessary, and nux vomica, quinine, and iron are valuable in many cases. If the heart-power fails, digitalis may be required, but its use should be postponed until the indications of failing compensation are clear. Sudden increase of the cyanosis may require venesection, but in all cases it will be safer to make trial of leeches first. A few large-sized leeches about the neck or chest may suffice to restore the cardiac power by relieving congestion, but if they fail of this end blood should be let directly from a vein.

DISEASES OF THE BLOOD-VESSELS.

BY WILLIAM PEPPER.

DISEASES OF THE ARTERIES.

ACUTE inflammatory lesions of the intima of the aorta, similar to those met with in the endocardium and upon the valves, may occur in association with the latter or as distinct lesions.

ACUTE AORTITIS.

Etiology.—In the majority of cases there is evidence of some general intoxication, since most cases are found after typhoid fever, diphtheria, pneumonia, or other infectious diseases. Gout, rheumatism, alcoholism, lead-poisoning, and other conditions have been cited as causes, but probably atheromatous ulcers have been mistaken for ulcerations of acute aortitis.

Morbid Anatomy.—The lesions observed are similar to those seen in endocarditis. Localized thickening of the intima, with a capping of fibrin deposited from the blood, is observed in some cases, while in others destruction and ulceration leads to an appearance like that of malignant endocarditis. Sometimes the fibrinous deposits are as large as a cherry or even larger, as was the case in a recent instance under my observation. In cases of endarterial ulceration acute aneurismal dilatation or rupture of the aorta may occur.

Symptomatology.—Pain in the region of the aortic arch is usually present. Sometimes it partakes of the nature of substernal soreness or tenderness; in other cases quite severe stabbing pain is experienced, and it may, according to Dobell, be referred to the right shoulder. Palpitation of the heart and throbbing of the vessels are generally noted, and may be prominent symptoms. Moderate fever is usually present, and there may be rigors or distinct chills. In the latter cases the suspicion of embolism is always aroused, since portions of the fibrinous excrescences are readily broken off and carried into the circulation. The embolic manifestations resulting are similar to those in endocarditis, and their severity will depend upon the nature of the inflammatory process in the aorta.

Diagnosis.—The diagnosis of acute aortitis cannot be established with certainty. The points which would distinguish it from endocarditis would be the greater diffusion and intensity of the thoracic pain and the absence of a murmur at the apex. A systolic murmur may, however, be present over the upper part of the sternum.

Prognosis.—The outlook is always grave, and fatal embolism or rupture of the aorta may occur at any time.

Treatment.—Rest, restriction of the diet, cold applications to the chest, and sedatives to quiet the action of the heart may be employed as in acute endocarditis. Calomel or potassium iodide may be administered, but probably have no power to cause absorption of the fibrinous deposit. In septic cases with fever and chills quinine and general stimulants should be employed.

ARTERIO-SCLEROSIS.

SYNONYMS.—Endarteritis chronica deformans (Virchow); Arterio-capillary fibrosis (Gull and Sutton); Atheroma.

Definition.—Arterio-sclerosis is a degenerative and inflammatory disease of the vascular system, with secondary fibroid changes in other ogans. The morbid changes may involve the arteries alone or may extend to the capillaries and veins as well. The aorta alone may be affected, but more commonly arterio-sclerosis is a general disease of the whole arterial system. It is difficult to determine how much of the disease is due to inflammatory and how much to degenerative changes, but probably in every case there is a mixture of both classes. Sclerotic changes in the internal organs and heart are rarely absent, and in many cases assume proportions which render the underlying arterial changes of secondary significance.

Etiology.—Arterio-sclerosis is essentially a disease of old age, the great majority of pronounced cases occurring in persons beyond forty years of age. It may result from the physiological processes of involution of advanced years, without any other determinable cause, and there is often a manifest hereditary tendency in certain families. The male sex is predisposed, because men are exposed to the causes of the disease more than women.

Toxic conditions of the blood are the most important etiological factors, and arterio-sclerosis plays a part in the morbid anatomy of chronic alcoholism, lead-poisoning, gout, syphilis, malaria, rheumatism, Bright's disease, and diabetes. In these cases the toxic agents may act directly and destructively, or may bring about the arterial changes by leading to contractions of the peripheral vessels and thereby increasing blood-pressure. In the case of alcoholics and those who habitually overeat there is repeated over-filling of the bloodvessels, which certain authors have regarded as the important etiological factor in the production of hypertension and subsequent sclerosis. In persons who have contracted syphilis arterial changes sometimes occur in early life, two of the most marked cases I have recently observed having occurred in young men respectively twenty-six and twenty-nine years old. Another cause of arterio-sclerosis in young men, and an important factor in cases at any age, is muscular exertion. As a result of the physical efforts involved in laborious occupations, such as mining, blacksmithing, and the like, there is impediment to the circulation and increased vascular tension, which gradually induces sclerotic changes in the arteries.

The relation of Bright's disease to arterial sclerosis is by no means a constant one. Unquestionably in certain cases primary disease of the kidneys leads to secondary arterial changes by causing increase of the blood-pressure or by direct action of toxic agents, whereas in other cases the renal lesion is consequent upon a general sclerosis of the arteries.

Occasionally arterio-sclerosis seems to be dependent upon infectious diseases, like typhoid fever, scarlet fever, and variola, or upon a cachectic state of the system, as is seen in cancer and tuberculosis.

Purely mechanical causes sometimes lead to localized forms of arterial disease, as I have recently seen in a case in which the pulmonary artery to its minutest subdivisions was highly sclerotic and atheromatous as a result of mitral stenosis. The aorta and the arteries of the systemic circulation were little affected.

Morbid Anatomy and Pathology.—The morbid changes of the vessels are of two kinds—the localized or nodular, and the diffuse—but in most cases there is a combination of the two.

The nodular form of sclerosis is most decided in the aorta, and may be unassociated with diffuse thickening of the aorta or with alterations of any kind in the smaller vessels. There are seen upon the inner lining of the artery grayish or yellowish elevations, which present an appearance not unlike that of cartilaginous plates. The edges are either abrupt or gradually sloping. In size these sclerotic plates vary from the smallest points to that of a large coin, and they may be so numerous that the intima of the aorta is everywhere irregular and roughened. The plates are generally most marked at the orifices of the coronary and other vessels, and may lead to serious obstruction to the circulation in these arteries. In the later stages degenerative or necrotic softening occurs within the plates, and they are then composed of soft, molecular material or débris, the lesion at this period receiving the name of "atheromatous abscess." The latter may rupture upon the surface of the intima, discharging its contents and giving place to the "atheromatous ulcer," an irregular, necrotic area, often covered with deposits of fibrin. The final change is calcification, which may occur in the base of the atheromatous ulcer or in the plates before their rupture. True ossification has occasionally been observed. In marked cases of atheroma of the aorta it is not unusual to find a combination of the various lesions, the intima being extremely rough and irregular in appearance, and presenting deposits of white thrombi upon the edges of the calcareous plates or in the fissures between them.

The same class of changes are also seen in the medium-sized and smaller arteries, though rarely as distinctly as in the aorta.

The media and adventitia may be thicker than normal, and the former may present more or less calcareous infiltration. In other cases, however, and particularly when localized dilatations of the vessel are present, the middle and outer tunics are thinned and degenerated.

Microscopically, the earliest change, as Köster pointed out, is infiltration surrounding the vasa vasorum of the media and adventitia. Subsequently the muscle-fibres of the media undergo hyaline and fatty degenerations. Following this, the subendothelial tissue of the intima proliferates and produces the sclerotic plates which are the striking macroscopic feature. These consist of dense sclerotic tissue, containing brightly-staining cells and often having a decided hyaline appearance. The subendothelial thickening may be looked upon as in some measure compensatory of the weakened condition of the media and adventitia, and as contributing to the preservation of the normal lumen of the vessel. Though these formations do project from the surface as nodules when the vessel is examined post-mortem, experiment has shown (Thoma) that under a pressure of mercury equal to the normal blood-pressure the nodule merely fills what would otherwise be a depression on the inner lining of the vessel. In the final stages, when degenerative changes have taken place, the atheromatous abscess is composed of granular débris, and may contain cholesterin plates and fat-needles.

The diffuse form of sclerosis, unassociated with the nodular, is more frequently met with in the smaller arteries than in the aorta. It is frequently seen in strongly-built, muscular men whose work has been laborious and who have been much exposed. In senile sclerosis there is also diffused thickening, though here nodular elevations are also present as a rule. The arteries are thickened and dilated, and at the same time lengthened, so that they become more or less tortuous. In an extreme instance I saw the brachial artery completely turned upon itself, so as to form a loop which could be distinctly seen under the skin. The intima presents spots of dull-white appearance, but not the distinct nodules before described. There may also be areas of degeneration or atheroma. Microscopically, the subendothelial tissue of the intima is greatly thickened, and the muscle-fibres in the media are variously degenerated. Sometimes the fibres are distinctly fatty or necrotic, at other times they are converted into hyaline material, and every resemblance to muscular tissue may be lost. In senile cases considerable calcareous deposition may occur, and the vessels may be converted into rigid tubes. The adventitia is thickened and dense.

In marked cases thickening of the capillaries is not uncommon, and there may be complete obliteration of their lumen in certain situations, as in the glomeruli of the kidneys. Obliterative endarteritis may also occur in the smaller arteries. The latter has been regarded as more characteristic of syphilitic sclerosis than of other forms. Involvement of the veins, or phlebosclerosis, is sometimes met with, especially in the lower extremities, but very rarely reaches high grades. Cases in which the arteries, capillaries, and veins are all involved have been very appropriately designated "angio-sclerosis."

Arterio-sclerosis is far more commonly present in the aorta than in other vessels. It is most marked in the ascending part of the arch, and diminishes in severity in the transverse portion, in the descending part, in the thoracic and abdominal aorta. The order of frequency with which other vessels are affected has been stated by Rokitansky to be—splenic, iliac, femoral, coronary, the cerebral vessels, the uterine, brachial, internal spermatic, common carotid, and hypogastric. The arteries of the stomach and the mesenteric vessels are rarely involved, and the pulmonary least commonly, excepting in cases of mitral disease or other conditions which impede the pulmonary circulation.

Associated Changes in the Vascular System and Internal Viscera.—In the early stage, when the elasticity of the aorta and other arteries has been impaired by degeneration of the media and adventitia, there is increased demand upon the heart and in consequence hypertrophy is commonly present. In senile cases, however, there is quite as often no enlargement, and there may even be brown atrophy. At the same time, if the coronary vessels are narrowed at their orifices or in their continuity, fibrous myocarditis is nearly always associated, and may contribute greatly to the increased size of the heart. In the later stages there is usually some fatty degeneration as well.

The weakened arteries are prone to undergo dilatation, with formation of aneurisms. The time when this occurs is limited, according to Thoma, to a period of about a year's duration, occurring at about the fortieth year of life, this date being that which precedes the compensatory thickening of the intima. After this time aneurisms are less apt to be developed. Miliary aneurisms are found especially in the brain, and may occasion cerebral hæmorrhage. Miliary aneurisms were found by Demance in four cases of disseminated cerebral and spinal sclerosis, and by Mendel in one of general paresis; and the arterio-sclerosis was regarded by both observers as an essential element in the pathology of the disease in question.

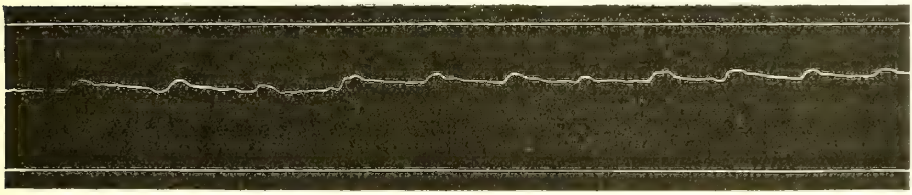
Sclerosis of the vessels and interstitial tissue of the kidney, the liver, and the pancreas is a most important part of the morbid anatomy. The kidney may be contracted and hard, with more or less adherent capsule, and on microscopic examination insterstitial overgrowth of fibrous tissue and sclerosis and hyaline degeneration of the glomerular and other vessels are observed. Analogous changes are seen in the liver, the pancreas and other organs.

Symptomatology.—Arterio-sclerosis may be a latent disease, remaining unsuspected until the lesions are discovered at the autopsy. In other cases the symptoms are so mild that little attention is paid to them, though the condition would be evident on careful examination.

No uniform clinical course can be sketched, for the symptoms of the disease vary widely according to the vessels involved. It is useful, therefore, to recognize certain types, such as the cardio-vascular, the renal or vaso-renal, and the cerebral forms.

Cardio-Vascular Type—The important condition in the early stages is the increased arterial tension, and upon this depend the various symptoms. The pulse at the wrist is peculiarly hard and incompressible. This is due to increase of the thickness of the wall, as well as to increased tension, but it may be difficult to estimate the amount of the latter on account of the rigidity of the artery. Sometimes in later stages, when calcification has become marked, the walls may be so stiffened that no pulse at all is palpable, but the artery may be felt as a rigid cord, or so irregularly thickened by nodules that the sensation imparted to the finger is likened to that of a bird's trachea. The character of the wave is slow and deliberate—the pulsus tardus. The rate is usually diminished, and a comparison of the time of the pulse with that of the apex-beat may show decided retardation of the wave, due, of course, to loss of arterial elasticity. The sphygmogram shows a gradual ascent with a broad top and sustained wave. The tidal wave is often very marked, and the dicrotic notch, as a rule, indistinct or obliterated (Fig. 27). The increase of arterial tension is due to the impediment to the circulation in the arteries and to increased action of the heart. The latter soon leads to hypertrophy, which may be recognized by increased dulness extending to the left and downward. On auscultation a decidedly accentuated, oft-times ringing, second sound is heard over the root of the aorta, and serves as an indication of the increased aortic tension. The first sound of the heart is comparatively weak, and when the ventricle is dilated or when myocardial degeneration has supervened, a systolic murmur is not rarely audible at the apex. The root of the aorta may be so much dilated as to occasion distinct dulness under the manubrium sterni, and the transverse portion of the arch may be elevated so much that it can be felt by pressing the finger backward and downward in the supra-sternal fossa. A feeling of constriction about the heart is not uncommon, and there may be repeated palpitations or attacks of angina pectoris. The latter occur, in cases in which the aortic arch alone is diseased as a result of dilatation or possibly from implication of Pacini's bodies, which have been found in the walls of the aorta; but angina pectoris is a serious symptom, more particularly, in the cases in which the coronary arteries are also involved, and in which fibrous myocarditis has been established. In beginning cases I have frequently noted slight substernal pains, with palpitation and dyspnœa coming on after exertion, and occasionally mild and irregular fever with rigors are observed.

FIG. 27.



Sphygmogram from a case of marked Arterio-sclerosis, in a man aged 60 years. Vertigo, headaches, mechanical angina.

When the hypertrophy of the ventricle is decided, there may be a conscious throbbing of the vessels and an uncomfortable feeling of fulness in the head. At the same time, ophthalmoscopic examination may reveal marked pulsation and sclerotic changes in the retinal arteries as well as dilatation of the veins, and whitish spots of degeneration in the retina itself. The arteries may be distinctly narrow in certain portions, and the walls may present areas of hyaline degeneration, or occasionally miliary aneurisms. In these cases, however, the cerebral vessels are, as a rule, coincidently diseased, and the cerebral type of the disease is apt to be noted.

Finally, in cardio-vascular cases the power of the heart grows inadequate as a result of increasing dilatation or myocarditis, and the circulation fails. The blood-pressure now becomes decreased, the pulse grows weak and small, and is often more frequent than normal, though bradycardia not rarely persists throughout. Functional cardiac murmurs may now be developed, or if the sclerotic changes have extended to the aortic valves, organic defects of the valve may be present. The functional murmur may be mitral or aortic, and is always regurgitant. Attacks of palpitation occur with greater frequency; the heart's action may be persistently irregular; and dyspnœa is developed on slight exertion. Syncopal attacks or constant faintness may be marked, and paroxysms of cardiac asthma sometimes assume a prominent place in the symptomatology. Emphysema and chronic bronchitis not rarely add to the respiratory difficulty, and congestions of the internal organs, as in valvular disease, give evidence of the failing circulation. A little dropsy in the feet is not uncommon, but anasarca is rarely observed. The urine, which during the stage of compensation and hypertension was copious, light-colored, and rarely albuminous, now becomes decreased in quantity and more constantly contains albumin.

At any stage portions of the white thrombi attached to the roughened intima may be broken off and carried as emboli to the various internal organs. In senile cases advancing endocarditis or occlusion of the peripheral vessels by emboli or thrombi may occasion gangrene of the extremities.

Vaso-renal Type.—The renal changes characteristic of arterio-sclerosis may be slowly developed in a case which has presented the symptoms of cardiac and arterial disease, or may occur without previous evidences of arterial disease. The quantity of the urine becomes greatly increased, the specific gravity is diminished, the color light, and a small quantity of albumin with hyaline casts is detected. Hypertrophy of the left ventricle and accentuation of the aortic second sound is noted, as in cases of primary renal disease. Dyspnœa and asthmatic seizures are not uncommon. It is often difficult, and in certain cases probably impossible, to determine whether the renal condition is secondary or primary to the vascular sclerosis. In some cases, however, the changes in the urine are gradually developed after the evidences of general arterial disease have become established, and in these the nature of the renal condition is apparent.

Cerebral Type.—In certain cases headache, tinnitus aurium, and vertigo are the only symptoms present throughout the disease. In others syncopal attacks or transitory palsies or aphasia evidence the disturbance of cerebral circulation. Symptoms of this kind may occur in persons of apparently robust health, but are especially marked and significant in the aged and infirm. Gradually degenerative changes in the brain are developed, and are indicated clinically by loss of memory, increasing weakness of intellect, and other symptoms of anæmic softening of the brain. Cerebral hæmorrhage may occur before degenerative changes have been established, but is more frequent subsequently. Transitory disturbances of vision are sometimes noted. The association of arterio-sclerosis with certain cases of locomotor ataxia, disseminated sclerosis, and general paresis has been observed.

Cirrhosis of the liver and pancreas are not often marked, and rarely occasion notable symptoms. The area of liver dulness may be decreased, and there may be congestion of the portal circulation ; but the hepatic symptoms are generally obscured by the more serious disturbances of the circulation and of the renal function.

Diagnosis.—Thickening of the peripheral arteries, either the radial, brachial, or temporal, hypertrophy of the heart, and increased arterial tension, with accentuation of the aortic second sound, are important points in the diagnosis. If all are present, the diagnosis may be positively asserted. The presence of arcus senilis is a confirmatory sign, though too much stress has probably been laid upon it, as I have seen the most marked arterio-sclerosis and atheroma unattended by the least haziness at the sclero-corneal junction.

The differentiation of arterio-sclerosis, with dilatation of the heart or fibrous myocarditis, from valvular disease may be impossible when the murmurs of relative incompetency of the valves are present; but with rest and cardiac tonics the heart may regain its power and the murmurs may disappear. In any case the treatment would be the same, and the diagnosis is therefore a matter more of scientific than of practical importance. The same may be said of the differentiation of primary renal contraction from that which is secondary to arterio-sclerosis.

Prognosis—When sclerotic changes have taken place in the arteries, there is probably never restoration of the normal condition, though Luzet asserts that he saw the thickening of the peripheral arteries disappear in a syphilitic case under specific treatment. When arterial tension is greatly increased, palpation of the peripheral arteries may so nearly simulate sclerosis of the walls that the single observation of Luzet is not free from doubt.

The prognosis, as far as life is concerned, is not unfavorable, for many persons live to advanced age in whom arterio-sclerosis began at a comparatively early period. The danger of renal and cardiac disease is always present, and embolism or cerebral hæmorrhage may cause sudden death at any time. A rare cause of death is rupture of the aorta at the seat of an atheromatous ulcer.

Treatment.—Much might possibly be done to prevent the disease if suitable hygienic and dietetic measures could be taken in advance, but this is, as a rule, manifestly impracticable. When recognized early, a quiet manner of life and the correction of any habits which are known to produce the disease would serve to limit the extension of the process; and when possible such regulations must be enforced. The diet should be plain and non-stimulating. Huchard has particularly emphasized the value of milk in cases in which the renal function is inadequately performed. When there is a distinct history of syphilis, the iodide of potassium should always be used, and even when the patient has not had syphilis this drug may be of value. It should be given in doses of three to ten grains three times daily, with occasional intermissions to prevent iodism. It is often valuable for the control of anginoid pains, even though it may have no power to cause absorption of the fibrous tissue in the vessel walls.

In the cases of subacute aortitis in which substernal pain, rigors, and slight fever are observed counter-irritation by blisters or the cautery over the sternum, and the internal use of quinine and iodide of potassium, constitute the most successful treatment associated with prolonged rest.

Increase of the arterial tension is best relieved by the use of nitro-glycerin or nitrite of sodium. In the later stages, when the heart-power fails, cardiac stimulants may be administered, but care must always be taken that the tension be not excessively increased by digitalis lest cerebral hæmorrhage result.

The treatment of the anginoid paroxysms is to be conducted in the manner laid down in the description of Angina Pectoris.

ANEURISM.

Definition.—The term "aneurism" is applied to more or less circumscribed dilatation of the arteries. By some authors the name is restricted to those forms alone in which a yielding or rupture of one or two coats of the artery has led to a localized dilatation, but writers generally include also dilatations which result from a gradual stretching of the whole wall, as well as to localized blood-sacs which communicate with an artery, but are surrounded by a wall derived from the neighboring tissues.

Etiology.—In all cases of aneurism some congenital or acquired weakness of the arterial wall leads to the development of the lesion. The most frequent cause is arterio-sclerosis, and it is particularly in the earlier stages of this disease, when degeneration of the media (Köster, Thoma) has occurred, and before compensatory thickening of the intima has supervened, that aneurism is apt to be developed. The conditions, therefore, which give rise to this disease are important in the etiology of aneurism. Syphilis, alcoholism, gout, rheumatism, and the severe muscular exertion entailed in laborious occupations are the important elements in the etiology of the primary arterial disease. Very frequently more than one of these factors is operative, as in the case of soldiers or laborers who have contracted syphilis and who have used alcohol excessively. Under these circumstances aneurism sometimes occurs in quite young men, but, as a rule, it is a disease of middle life, occurring with greatest frequency from the ages of thirty-five to fifty years. Cases have occasionally been reported in children, as that of Moutard-Martin in a child of two years, and that of Roger at ten years. The male sex is affected with much greater frequency than the female sex, probably because the causes of arterial disease are more commonly present in the former.

The immediate determining cause of the aneurism is frequently some unusual strain, which raises the blood-pressure, as in lifting or mounting steep elevations. Bronchitis or other conditions which induce paroxysms of coughing act in a similar manner, and I have recently seen an aneurism suddenly develop in a patient from straining at stool. Traumatism sometimes seems to be the immediate cause, as in patients who trace the symptoms to a fall or blow upon the chest.

Heredity is a factor of some importance. Lancisi, for example, records the occurrence of aneurism in a grandfather, father, and son. In these cases there may be some congenital defect in the arterial walls or a tendency to arterial disease. Occasionally congenital aneurisms are observed, as in the remarkable case of Phenomenow, in which a large aneurism of the abdominal aorta formed an obstruction to labor. The periarteritis nodosa of Küssmaul and Maier has also been regarded as a form of congenital aneurism.

Aneurisms are sometimes formed by traction in young children or during fœtal life. This is seen especially at the origin of the ductus Botalli, where conical dilatation of the aorta is not infrequent.

Embolism may lead to aneurismal dilatation by causing local degeneration or injury of the vessel-wall. In the case of calcareous particles from the heart-valves a purely mechanical injury causes local weakening of the arterial wall, but in case of septic emboli, as in ulcerative endocarditis, acute inflammatory lesions of the intima are first developed.

Morbid Anatomy.—Anatomically, aneurisms have been classified according to their shape, as fusiform, cylindrical, or cirsoid when there is a gradual dilatation or ectasy; and as saccular when a localized area of weakness has given rise to a circumscribed dilatation communicating with the vessel by a well-defined orifice. The term dissecting aneurism has been applied to cases in which a partial rupture within the vessel allows the blood to find its way between the tunics. Such aneurisms are not infrequent in the arch of the aorta and in the smaller vessels of the brain. A remarkable instance was found postmortem in one of my cases at the University Hospital, in which neither the history nor the physical signs indicated the existence of aneurism. There was a transverse rupture of the intima and media near the aortic valves, from which the blood had gradually found its way along the entire length of the aorta, and by secondary ruptures into the aorta just above the bifurcation, and into one of the iliac arteries. There were practically two aortas side by side, separated by a narrow partition.

Sometimes arteries communicate with veins and form the so-called arteriovenous aneurisms. If there be an intervening sac, the name varicose aneurism is applied; but if the communication be direct the condition is spoken of as aneurismal varix.

The terms true and false aneurism have been used so variously by different authors that little value now attaches to them.

The commonest forms of aneurism are the saccular and the fusiform. In the former there is a localized sac-like dilatation, which is sometimes irregular in outline, having secondary dilatations in its wall, and which communicates with the artery, laterally, by a more or less constricted orifice. The wall of the aneurism is composed of the thickened intima and adventitia, which have gradually yielded before the blood-pressure at a point where the degenerated media had ruptured. There may be no trace of the media in the aneurismal wall at any point, but especially is the latter absent at the summit of the sac. The intima is thickened, and may be more or less degenerated and calcified in certain areas; the adventitia is also thickened; but finally all the coats become thinned and the aneurism ruptures. The surrounding tissues may now form an adventitious covering, as in cases in which thoracic aneurisms perforate the chest-walls, the fasciæ and muscles forming a restraining wall, or in cases of rupture into the lung, when the pulmonary substance may be completely destroyed, leaving a cavity enclosed by the thickened pleura in communication with the original sac.

The cavity of the aneurism usually contains clots of blood, and frequently these are laminated and partly organized. The formation of clots within the sac is a conservative process which sometimes leads to obliteration of the cavity and thus to spontaneous cure; but at any time vessels springing from the main artery at the seat of aneurism may be occluded, or parts of the clot may be carried into the circulation as emboli. Degenerative changes may occur within the clots—either softening, the coagula breaking down and forming a chocolate colored fluid, or calcareous infiltration.

In the case of fusiform and other ectatic aneurisms all three coats of the vessel may be preserved in the aneurismal wall and may be greatly stretched, but even in this form of aneurism the media is deficient in places. Finally, yielding may take place and rupture may ensue, as in the saccular form. Laminated or other clots are less frequent than in saccular aneurisms.

The changes induced by aneurisms upon neighboring structures by pressure will be considered below. The amount of pressure and of disease of surrounding parts will of course depend upon the size of the aneurism. The latter may vary from the microscopic miliary aneurisms of cerebral vessels to large tumors of the size of a child's head or larger. There is usually but one aneurism, but not rarely several are observed in the same vessel or in different vessels.

Aneurism of the Thoracic Aorta.

Aneurisms are far more commonly met with in the thoracic than in the abdominal aorta or in other vessels. Of 234 cases of aortic aneurism collected by Crisp, the thoracic portion was affected in 175 (74.8 per cent.) and the abdominal in 59 (25.2 per cent.). Any part of the vessel from the aortic valves to the diaphragm may be involved, but the frequency diminishes as the distance from the heart increases. Over one-half are found in the ascending portion, somewhat more than a quarter in the transverse part of the arch, and about one-eighth of the cases affect the descending portion. Not rarely small aneurismal dilatations spring from the sinuses of Valsalva, and very rarely, as in the case of Bramwell, a large aneurism may have this as its point of origin. In the ascending portion the aneurism sometimes reaches enormous size, and may eventually perforate the anterior wall of the chest and appear as an external tumor of the size of half a cocoanut. In this situation the saccular form is most common. In the transverse portion fusiform dilatations are more frequent than in the ascending part of the arch, and the origin of the innominate, right carotid, or subclavian, or all of these, may be involved. The exact position of the aneurism on the vessel is determined to a large extent by the direction of the blood-current and the points where it strikes most forcibly against the wall. Thus, in those of the ascending and transverse parts of the arch the aneurism is generally upon the anterior or convex surface, while in those of the descending aorta aneurisms more commonly develop posteriorly. Very frequently there are several smaller in association with one large aneurism, or the original sac may have secondary pouchings at different portions.

Symptomatology.—The symptoms of thoracic aneurism are largely the result of pressure upon neighboring structures, and therefore depend upon the situation of the aneurism and upon its size. The severest symptoms, as a rule, occur in those aneurisms which involve the transverse portion of the arch, because in this situation the narrowness of the antero-posterior diameter of the chest leads to great compression of the soft parts as soon as the aneurism reaches considerable size. In cases of aneurism of the ascending and descending portions the sac may reach notable proportions before any signs of intrathoracic pressure are observed, but of course this state of affairs is exceptional. Latency, however, is not unusual in case of small aneurisms, as in those of the sinuses of Valsalva, and the patient may die suddenly of rupture without ever having presented any symptoms.

One of the earliest and throughout the case one of the most urgent symptoms is pain. Occasionally, when the aneurism is suddenly developed as a result of severe straining efforts, a sharp pain in the upper portion of the chest may denote the rupture which has taken place in the tunica media, and the patient may experience the sensation of a "giving way." In the later stages pain is due to stretching of the fine nerve-filaments in the wall of the aorta itself, or to pressure upon surrounding structures. It is usually more or less constant, but is subject to severe exacerbations when the blood-pressure is elevated and the sac becomes distended. The patient's features become drawn and present the lines denoting constant suffering and anxiety. When the sac reaches the chest-walls and begins to cause erosion of the bones, the pain becomes of an unremitting, dull, boring character. This is especially prominent in cases in which the descending aorta is involved and erosion of the vertebræ is taking place. It is remarkable, however, what severe pressure may in some cases be exerted without inducing the least pain. In a recent case under my observation an aneurism of the ascending arch of the aorta had eroded the sternum, appeared externally as a large tumor, and finally ruptured on the surface, without having caused any decided subjective sensation excepting dyspnœa ; and in another case now under treatment the tumor has become of such size as to cause dulness over a large part of the thorax to the right of the sternum, and yet the man has never experienced any pain. In some cases, and especially in those near the heart, severe anginoid paroxysms may be experienced, and the aneurism may in other respects remain latent until death occurs. When pressure is exerted upon the large nerve-trunks, the pain is more or less shooting and lancinating in character, and may be referred to distant parts. Not infrequently, in aneurisms of the transverse portion of the arch, there are severe neuralgic pains passing along the nerves of the left arm or to the neck and occiput. In those of the descending aorta the intercostal nerves may be pressed upon, and in cases in which the aneurism is situated just above the diaphragm the case may be regarded as one of intercostal neuralgia or of lumbago, the aneurism being wholly overlooked. Accompanying the pain in these cases there is not rarely some anæsthesia of the skin and loss of power in the distribution of the nerve which is pressed upon.

Disturbances of respiration are rarely absent in cases of large aneurisms and may be most urgent even in cases of small ones which are so situated as to exercise injurious pressure. Dyspnœa may be due to pressure upon the lungs, the trachea, or bronchi, or upon the nerve-trunks. In case of pressure upon the lungs, as is seen in aneurisms upon the convexity of the ascending aorta when the aneurism compresses the right lung, there may be but slight dyspnœa until the sac reaches a large size. In case of very large tumors the lung-substance may be considerably consolidated, and the physical examination shows the collapsed condition of the vesicular substance. More serious dyspnœa is occasioned by pressure on the trachea or bronchi, and even a small aneurism may give rise to the extremest grade of orthopnœa if its situation be such that one of the bronchi is compressed. The breathing is noisy and stridulous, and there is a loud, ringing cough, with copious watery or later mucopurulent expectoration. Physical examination will reveal a decided diminution in the expansion of one side, with weak or almost absent breath-sounds and with diminished tactile fremitus. Sibilant or sonorous râles are commonly heard, and may obscure all other sounds. These signs are sometimes of the utmost value in the detection of a deep-seated aneurism which does not reveal itself by the ordinary physical signs. In cases of this kind there is not unusually some fever from retention of the secretions, and in extreme instances there may be bronchiectasis and consolidation of the lung-substance with suppurative or gangrenous softening. The latter conditions have doubtless been mistaken for tuberculosis in some cases, as in the collected cases of Hanot pulmonary tuberculosis was said to have occurred in 18 of 42 cases. The sputa not infrequently contain a little blood which may come from the soft granulations and ulcerations which are formed in the bronchus at the point of pressure, or by gradual filtration from the aneurism itself. Decided hæmoptysis denotes the rupture of the aneurism into the lung-substance or into the trachea or bronchi, and generally proves fatal. In some instances, however, the patient is relieved by the loss of blood and may survive repeated attacks, losing considerable quantities of blood in each. Paroxysms of sudden and severe dyspnœa are occasionally observed in cases of aneurisms of the transverse portions of the arch, and are referred by Bristowe to sudden increase of pressure upon the trachea when the sac becomes distended. In some instances it has been possible to demonstrate this by laryngoscopic and tracheoscopic examination; but there is danger of mistaking the slight normal pulsation visible in the trachea (Türck; Gerhardt) with pathological conditions. Other observers refer the paroxysms of dyspnœa to spasm or paralysis of the larynx from pressure on the recurrent laryngeal nerve.

Asthmatic seizures occasionally result from implication of branches of the vagus, and may be associated with attacks of angina pectoris or with vomiting and other gastric symptoms if the cardiac filaments on the one hand or the gastric branches on the other hand are involved.

Laryngeal symptoms are frequently observed in aneurisms of the arch of the aorta, and result from pressure upon the recurrent laryngeal nerve. That of the left side is the more commonly involved, but not rarely both are affected. In the early stages there may be attacks of spasm of the larynx, causing dyspnœa or stridor, but soon the vocal cord of one or both sides becomes paralyzed and immobile. The voice is husky or whispering, and when both vocal cords are paralyzed there may be complete aphonia. The cough is loud, hard, and clanging, and not rarely is distinctly paroxysmal. The most marked laryngeal symptoms occur when paralytic contractures have supervened. Paralysis of one of the vocal cords is sometimes unattended with changes in the voice or any other symptoms.

Alterations of the pupil and vasomotor disturbances of the head and neck result from pressure upon the sympathetic nerve or ganglia. In the early stages, when the nerve is merely irritated by the pressure, the pupil on the affected side is dilated and the skin may be paler than normal. Later the nerve becomes degenerated and paralyzed, and contraction of the pupil with flushing and sweating is then observed. All of these symptoms are most frequently observed in aneurisms of the transverse part of the arch, but the vasomotor changes are much less common than pupillary alterations.

Swallowing may be interfered with by pressure upon the œsophagus or by reflex contractions of its walls, and there may accordingly be constant or paroxysmal dysphagia, with gradual emaciation. Pressure upon the œsophagus is especially common in aneurisms of the descending aorta opposite the eighth dorsal vertebræ, but also occurs in cases in which the transverse portion of the arch is involved. The simulation of cancer of the œsophagus may in some cases be very close, and the physician may be tempted to pass the sound. In all cases, however, this procedure must be avoided until aneurism has been most carefully excluded.

Circulatory disturbances may result from pressure upon the veins or from failure of the heart-power. In the early stages attacks of palpitation are not unusual, and may occur alone or in combination with seizures of angina or of paroxysmal dyspnœa. In the final stages the cardiac power may fail, and various indications of disturbance of the peripheral circulation may develop. Large aneurisms not infrequently compress the superior or inferior cava, the innominate, or other veins, causing congestion, cyanosis, and œdema of the lower part of the body, of the upper half, or of one or the other arm respectively. When the inferior cava is compressed the legs and abdominal walls are congested and dropsical, and there may be ascites, with congestion of the internal organs and albuminuria. In case of compression of the superior cava cyanosis is observed in the upper part of the chest, of the head, and of the arms; and, if the azygos vein be simultaneously compressed, the whole of the chest may be cyanosed. There are often spongy masses of tissue above the clavicles, to which the term "collar of flesh" has been applied; and there may be decided exophthalmos. Headache, somnolence, and other symptoms of disturbance of the cerebral circulation are not unusual.

Aneurisms of the descending aorta may cause erosion of the bodies of the vertebræ at the point of compression, with paræsthesia, or loss of power, in the legs.

The clinical course of aneurism of the thoracic aorta is extremely varied and may be greatly modified by complications. When the aneurism is situated at the beginning of the ascending portion, pericarditis not uncommonly results, and it is to a degree conservative in nature, for the resulting adhesions prevent the early rupture of the sac which is to be dreaded in these cases. When the sac presses upon the lungs, more or less extensive pleural adhesions may be formed or destructive lesions of the lung itself result. Pneumonia may supervene, and is apt to be fatal, and phthisis is not infrequently observed. Thickening and retraction of the aortic valves with regurgitation may be present, and may seriously aggravate the symptoms. In these cases cerebral symptoms are especially common, and the patient may die suddenly from syncope or from paralytic arrest of the heart's action.

The appearance of the patient and his general condition are often most deceptive. For a considerable time there may be not the least external indication of disease, but when pain becomes marked, the features of the patient are drawn and the expression is anxious. Profound emaciation occurs when the œsophagus is obstructed or, as I have observed in rare cases, when the thoracic duct is compressed. Fever occurs in cases in which the lungs or bronchi are pressed upon, and may assume a markedly irregular type, with occasional rigors and chills. In a case of this kind recently under my care the appearance of the patient, together with the fever, chills, and sweats, indicated a septic process in striking manner. At autopsy a small aneurism, no larger than a walnut, was found compressing the left bronchus, and within this tube there were collections of putrid secretions.

Physical Signs.—The physical signs may be very obscure when the aneurism is small and deep-seated or covered by emphysematous lungs. In these cases a careful physical examination of the chest may reveal partial indications that might lead to a probable diagnosis; but in some instances thoracic aneurisms are latent from the complete absence of physical signs as well as symptoms.

Inspection.—A most important indication of aneurism is the appearance of a pulsating tumor of the chest. In the earlier stages there may be no protrusion, and yet careful inspection from the side may reveal an evident pulsation with each contraction of the heart. Later large tumors may result from erosion and bulging of the ribs or from perforation of the chest-wall. The commonest seat of pulsation is in the second and third interspaces to the right of the sternum, and in these cases, as a rule, the aneurism will be found to spring from the ascending arch. When the transverse portion is involved, the sac projects against the manubrium or to the left of it, and there may sometimes be visible pulsation in the suprasternal fossa. An aneurism of the descending portion of the aorta may be visible as a pulsating tumor situated to the left of the spinal column, between it and the scapula. Pulsation above the second rib and extending to the neck on the right side occurs in cases in which the innominate artery is involved. The skin over the external tumor is smooth and glistening, and when external rupture is threatened usually becomes infiltrated with blood or of gangrenous appearance.

The apex-beat of the heart is usually more or less displaced to the left and downward, and is frequently seen in the sixth interspace outside the nipple line. In cases in which valvular lesions coexist with aneurism this may be partly due to hypertrophy and dilatation of the heart, but in uncomplicated aneurism it is more commonly the result of displacement than of enlargement of the organ.

Palpation.—On palpation the pulsation of an aneurism is found to be strong, heaving, and expansile. The latter quality is marked in cases in which the external protuberance can be grasped by the hand, and may be at once evident by the separation of the fingers of the palpating hand at each systolic distention of the sac. When the tumor cannot be grasped, the expansile character may sometimes be demonstrated by placing pieces of moistened paper edge to edge upon the skin, when the papers are seen to separate a little with each pulsation. The expansile pulsation of aneurisms may be lost, as may indeed all pulsation, if the sac be filled with laminated clots.

In cases of aneurism of the transverse arch palpation in the suprasternal fossa may reveal the presence of an aneurism situated behind the manubrium.

In addition to the systolic pulsation there is sometimes a sharp diastolic shock which is of considerable diagnostic value. By auscultation this is found to be synchronous with the closure of the aortic valves, and is most evident in aneurisms situated on the root of the aorta. A systolic thrill is frequently noted, and is particularly common when there is general dilatation of the vessel, though it is not unusual in saccular aneurisms.

Percussion.—A localized area of dulness to the right of the sternum and above the third rib is always a valuable physical sign. Deep-seated aneurisms may give rise to no changes on percussion, but those which reach the surface of the chest cause a peculiar flatness and resistance to the finger quite unlike those noted in localized consolidations of the lung. The area of dulness, however, by no means indicates the size of the aneurism, for the lungs may be emphysematous and leave but a small part of the tumor exposed.

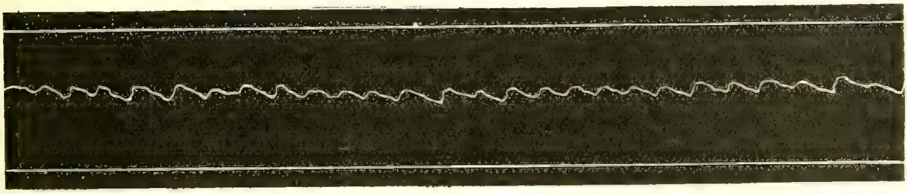
Auscultation.—The most characteristic sign of aneurism is a soft systolic murmur audible over the tumor and sometimes in the carotid vessels of both sides, and which results from the liquid vein generated in the passage of blood from the small vessel into the larger aneurismal sac. Conditions of blood-pressure may noticeably affect the murmur, so that it disappears and reappears from time to time. If the aneurism be filled with blood-clots, the bruit may disappear, and in some cases it is never heard. Aneurisms near the aortic valves frequently gives rise to a diastolic murmur in addition to the systolic, when the valves have become incompetent from over-stretching of the orifice or from secondary disease of the valve-segments. Probably in some cases the diastolic murmur may be due to regurgitation from the vessel beyond the aneurism into the sac, but this is certainly of rare occurrence. The murmurs generated in an aneurism are sometimes very loud, as in a recent case under my observation in which a loud to-and-fro murmur was audible at a distance of six feet from the patient.

A sign of considerable value, especially in deep-seated aneurisms and in those in which the murmur is absent, is accentuation of the aortic second sound. This is particularly noticeable in aneurisms situated near the origin of the aorta. When, however, relative or organic regurgitation supervenes, the second sound may be wholly absent. A faint and toneless first sound is heard in cases of aneurisms which have come to the surface of the chest or produced an external tumor. It is synchronous with the palpable systolic shock, and probably results from this.

Drummond of Newcastle has called attention to a systolic murmur heard over the trachea, and probably due to expulsion of air at each distention of the sac.

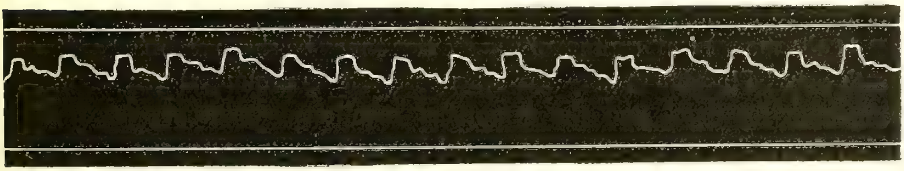
The pulse in cases of thoracic aneurism sometimes gives valuable indications of the existence or seat of the disease. If there be a large aneurism of the ascending part of the arch, a comparison of the time of the pulse at the wrist, in the carotid, the abdominal aorta, and femoral vessels with that of the apex-beat or the systolic pulsation of the tumor often shows a considerable delay or retardation. This is of course explained by the mechanical slowing of the current which results from the size of the aneurismal sac. At the same time, it may be noted that the pulses are all weaker than normal, and in the abdominal aorta or femorals there may be complete disappearance. When the aneurism is situated in the tranverse portion of the arch, the innominate artery being uninvolved, the pulses in the right arm and on the right side of the neck may be strong and almost synchronous with the apex-beat, whereas that of the left side and of the lower part of the body are decidedly weak and retarded. Still greater alterations in individual pulses may result from occlusion of the orifices of certain vessels in the aneurism by clots, by stretching of the sac, or by atheromatous changes. In every case, therefore, the character of the pulse in all of the accessible vessels should be ascertained, when deductions as to the exact location of the aneurism may be possible. The sphygmographic tracings are greatly altered by interposition of an aneurism between the heart and the peripheral artery, but no constant features are observable. The changes which are most characteristic are a marked obliquity of the up-stroke and obliteration of the secondary waves on the descending line. The wave, as a rule, is small, and the height of the curve is therefore reduced (Fig. 28). A comparison of the sphygmogram of the two radials may show on one side a normal curve and on the other side such a tracing as has been described, but too much weight must not be attached to this means of investigation. (See Fig. 29.)

Fig. 28.

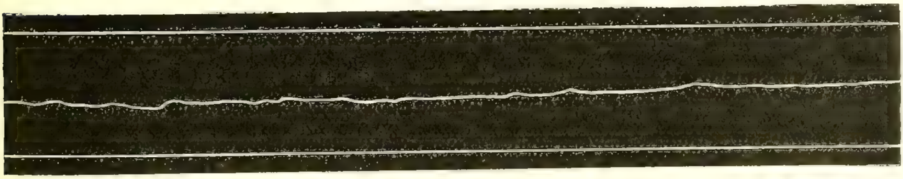


Sphygmogram from the Right Radial Artery, in a case of Aneurism of the Ascending Portion of the Arch of the Aorta.

Fig. 29.



Right Radial Pulse.



Left Radial Pulse.

Sphygmograms of the Radial Pulse on the right and left side, from a case of Aneurism of the Transverse Part of the Arch of the Aorta.

Ophthalmoscopic examination may show marked pulsation of the retinal arteries on the affected side, and little or none at all on the opposite side. In cases of large aneurisms with aortic insufficiency, but to a less extent without this, the capillary pulse of Quincke is sometimes observed. Decreased amplitude or even extinction of the pulse-wave during inspiration (pulsus paradoxus) is occasionally noted in the arteries whose parent trunk springs from an aneurismal sac.

Marked distention of the veins of the neck is noted in many cases when the aneurism reaches considerable size; and there may be decided undulation. If the inferior vena cava is pressed upon, distention of the veins of the abdominal walls and of the legs may be observed.

Tracheal tugging is a sign of considerable value which was pointed out by Surgeon-Major Oliver. To obtain this sign the patient is made to sit or stand upright with the chin depressed so as to relax the tissues of the neck. The cricoid cartilage is then grasped between the thumb and forefinger and pushed upward so as to stretch the trachea, when a downward dragging or tugging will be felt at each systole of the heart. This tracheal sign is most marked when the aneurism presses downward and backward against the left bronchus, but may occur when it has other situations and is attached to the trachea or neighboring parts. Sometimes the downward movement is so marked as to be readily visible on inspection of the patient's neck.

Terminations.—The tendency in all cases of aneurism is to gradual increase of the sac until some portion becomes too weak to withstand the blood pressure, when rupture takes place and death ensues from hæmorrhage. Before this time, however, death may occur from gradual exhaustion, or suddenly from heart failure, the latter being particularly common when aortic regurgitation has supervened. The fatal termination may also be determined by intercurrent affections and complications such as pleurisy, pericarditis, pneumonia, or embolism. Only in rare instances does deposition of blood-clots within the sac lead to spontaneous cure of the aneurism.

At the moment of rupture of the sac there may be sharp stabbing pain, and death may be instantaneous. In other cases life is prolonged for some time, sometimes for days, and the symptoms manifested—syncope, rapid, weak pulse, depression of the temperature, cold sweats—give evidence of the occurrence of large hæmorrhage. In rare instances the patient may recover, and survive several similar attacks.

The position of the rupture and the resulting physical signs will depend upon the seat of the aneurism. In those springing from the aorta just above the valves rupture into the pericardial sac is not unusual, and if death be not too sudden is attended by increase of the area of præcordial dulness, disappearance of the apex-beat, and muffling of the heart-sounds. Aneurisms of the ascending portion of the aorta may extend to the right and rupture into the right pleural cavity, and those of the descending aorta may perforate the left pleura, the physical signs in either case indicating fluid in the pleural cavity. Rupture into the bronchi or trachea or into the lung-structure is attended by violent hæmoptysis, and is usually followed by rapid death. The fatal perforation and hæmorrhage are, however, usually presaged by slight tinging of the sputum with blood, which comes from the granulations or ulceration of the trachea or bronchi at the point of pressure or by gradual filtration from the aneurism itself. Large aneurisms which have perforated the chest-wall and become visible as external tumors may occasion gangrenous sloughing of the skin and rupture externally. In some instances of this kind the blood may trickle from the point of threatened rupture for a long time before the fatal perforation ensues. Rupture into the œsophagus may be recognized by the vomiting of blood and by the appearance of bloody evacuations.

Very rarely the aneurism may perforate into the venæ cavæ, the pulmonary artery, or the auricular cavities of the heart itself. A remarkable instance of communication between an aneurism of the ascending aorta and the superior vena cava occurred in my service at the University Hospital some years since, and in the report made by Dr. Griffith and myself 28 similar cases were cited from medical literature. The condition is easily recognized when there are suddenly developed congestion and œdema of the upper part of the body, and when signs of aneurism, with a continuous murmur growing louder at each systole, are detected by physical examination. Rupture into the heart or into the pulmonary artery is generally quickly fatal, but life may be preserved for some time.

Diagnosis.—Two problems are presented in the diagnosis of aneurism of the thoracic aorta: first, the determination of its existence, and, second, the exact point of origin.

The first point may present little difficulty when the aneurism is large and there are distinct pulsation and tumor, but offers great difficulty if the aneurism be small and deep-seated. Aneurisms of the first part of the aorta, which not infrequently spring from the sinuses of Valsalva, are especially apt to remain latent, and are therefore easily overlooked. In other cases the symptoms present are so indefinite in nature that the existence of aneurism is not determined even after the most careful examination.

Obscure thoracic pain, anginoid seizures, and neuralgias radiating along the brachial plexus or the lower intercostal nerves should always give rise to suspicion, and should lead to careful examination. The points of value in determining the diagnosis are circumscribed area of dulness in the region of the aorta, pulsation and thrill over the same area, accentuation of the second sound, systolic murmur, and changes in the character or time of the pulses. Any one of these signs may be present without aneurism, but the combination is strongly indicative of this lesion. Tracheal tugging is an additional sign of value, but is frequently absent.

In another class of cases in which the diagnosis is obscure marked dyspnœa and cough, with signs of bronchial irritation, are prominent symptoms from the beginning. I have before referred to a case of this kind which terminated fatally, and in which a small aneurism no larger than a walnut was found compressing the left bronchus, into which it had ruptured. In such cases a careful comparative examination of the respiratory sounds and vocal fremitus on the two sides, with laryngoscopic and tracheoscopic examination, is always of value, and may establish the diagnosis.

The conditions with which thoracic aneurism is most apt to be confounded are solid tumors, simple abnormal pulsation of the aorta, and pulsating empyema.

The tumors most likely to simulate aneurism are glandular enlargements and sarcomatous or cancerous growths of the mediastinum, and in certain cases the diagnosis is practically impossible. If pulsation be marked, it will be noted that in tumor this is quick, and not deliberate nor expansile as in aneurism, and the force is rarely as great. The auscultatory phenomena may be simulated in tumor, but the systolic murmur is rarely as pronounced and the decided accentuation of the second sound is nearly always wanting. The diastolic shock on palpation and tracheal tugging are also absent; and pain is more commonly present in aneurism than in tumor. The history and general condition of the patient are also of value in arriving at a diagnosis. Aneurism occurs most frequently in persons from thirty-five to fifty years of age, whose labor has been severe, or who have had syphilis, but the general health is often little affected; tumor, on the other hand, is more common before the age of forty years, the history of syphilis is no more frequent than among people in general, and cachexia and secondary nodules in the axillary or cervical glands are frequently observed.

Marked pulsation in the aorta may be present in aortic regurgitation, in cases in which the margin of the right lung is retracted, and in spinal curvature with displacement of the aorta. In each of these the diagnosis is often difficult, though, as a rule, symptoms indicative of pressure and pain are less marked, while the auscultatory signs and the characters of the pulse observed in aneurism are absent. Simple dynamic pulsation of the aorta, of neurotic origin, seems to have been present in the case of Murray of Newcastle-on-Tyne, which Bramwell reports, but this condition is so rare that it need scarcely enter into a discussion of the diagnosis.

Empyema may occasion an external tumor near the heart from which it receives communicated pulsation. The impulse, however, is not so clearly circumscribed, nor so forcible and expansile as in aneurism, nor is it attended with the auscultatory phenomena met with in the latter. Pressure symptoms and the altered character of the pulse are wanting, whereas the signs and symptoms of pleural effusion are commonly present.

The diagnosis of the position of the aneurism will depend largely upon the point of pulsation, upon the character of the various pulses, and upon the associated symptoms.

Aneurisms of the ascending part of the arch grow to the right or forward, and commonly cause pulsation above the third rib and to the right of the sternum. The right recurrent laryngeal nerve may be compressed, and the expansion of the right lung may be greatly deficient. If the orifice of the innominate artery be involved, the pulse at the right wrist may be greatly altered. In rare cases the aneurism may project forward and downward over the origin of the pulmonary artery, and may be mistaken for aneurism of the latter, as in the case of Huber. Aneurisms involving the innominate artery alone are recognized by the existence of pulsation and tumor extending far above the clavicle, by the great alteration of the right pulse and by the normal character of the left.

Aneurisms of the transverse portion of the aorta project anteriorly or upward. The upper portion of the sternum may be pushed forward and pulsate, or the tumor may be felt in the suprasternal fossa. If the innominate artery be not involved, the right-radical pulse may be normal, whereas the left radial and the femoral pulses are greatly retarded and weakened. Pressure symptoms are particularly frequent.

Aneurisms of the descending portion of the thoracic aorta frequently remain obscure until tumor and pulsation are detected to the left of the spinal column. The retardation of the femoral pulse and the physical signs determined in the examination of the lungs and of the circulatory system are of great importance. Cases of indefinite obstruction of the œsophagus and of obscure neuralgic pains in the back or of obstinate intercostal neuralgia, should always lead to a careful examination.

Prognosis.—The outlook in aneurism of the thoracic aorta is always most grave. Recovery may take place spontaneously or under treatment, but can rarely be expected. Rupture may occur at any moment, or sudden death may result from other causes. Lebert analyzed a large number of cases and found the duration of life from the time of the first distinct symptoms to be about fifteen to eighteen months. Sacculated aneurisms of the ascending part of the arch are more favorable for treatment, and are of longer duration than are those of the transverse or descending portions, which are more frequently ectatic. Cases are recorded in which the patient survived for as much as ten years after an external tumor had appeared, but such are highly exceptional. In one truly extraordinary instance a letter-carrier pursued his arduous work for years, although presenting the most enormous aneurism of the descending aorta I have ever observed. When pressure is exerted upon the trachea, the bronchi, or the œsophagus, a fatal termination may be expected within a very short time—a few weeks or months. The condition of the general health and the nature of the patient's occupation will determine the length of life to a very great extent.

Treatment.—The object of treatment is to promote clotting within the aneurism and subsequent contraction of the sac. Conditions, therefore, which favor coagulation are to be induced. These are reduction of the quantity and pressure of the blood, slowing of the blood-stream, and increased coagulability of the blood.

The method of treatment suggested by Tufnell, and known by his name, favors the development of the conditions named, and is certainly of value in prolonging life if not in establishing a permanent cure. The essential elements in Tufnell's treatment are rest and restriction of the diet. The patient should be placed absolutely at rest, and should make as little mental or physical exertion as possible. By these means the cardiac pulsation and the blood-pressure are considerably reduced, but if the patient cannot lie in bed, he must be kept as nearly free from exertion as possible in some other posture. The diet should be rigidly restricted, especially the liquid portions. Tufnell advised two ounces of bread and butter and two ounces of milk for breakfast; two or three ounces of bread, and two or three ounces of meat, with two to four ounces of milk or claret, for dinner; and two ounces of bread and two ounces of milk for supper; but it will be found in practice that few patients will submit to so radical a reduction of food. A somewhat more liberal allowance of solids and drink must therefore be admitted. The effect of this form of diet is the diminution of the quantity of the blood, and probably an increase of its specific gravity and coagulability. Rest with restriction of diet certainly offers some hope of obliteration in cases of saccular aneurism, but is practically difficult to carry out. Small doses of morphine or other sedatives may be used at the same time to quiet the patient or to control pain, but for the latter symptom particularly the iodide of potassium is a remedy of surprising power. It should be given from the start in doses of five or ten grains three times daily, and may be increased to twenty or thirty grains, but, as a rule, the latter quantities are unnecessary. The manner of action is doubtful. Probably, however, it is by reduction of the blood-pressure with stimulation of the excretions that the drug exercises its power.

Systematic bloodletting is another adjunct of great value in addition to Tufnell's treatment. Six or ten ounces of blood may be abstracted from the veins of the arms every ten days or two weeks, provided the patient does not become excessively anæmic. The effect of bleeding is to decrease the blood-pressure and to increase the coagulability of the blood, results which would manifestly be offset or prevented by hydræmia should this follow the loss of blood. Bleeding as a curative measure is therefore of greatest value, or perhaps of any value only, in the early stages when the general health of the patient has been little impaired.

Various forms of local treatment have been advised, and sometimes good results have been obtained. Even deligation of the carotid or of the subclavian artery has been tried, but it is of questionable propriety. The insertion of horse-hair, fine catgut, or wire, galvano-puncture, injection of styptics, and the combination of electrolysis with insertion of wire have all been advocated, and may be resorted to when fair trial has been made of rest, restriction of diet, and potassium iodide. In the operation of galvano-puncture a needle insulated to near the point is introduced into the sac and attached to the positive pole of the battery. The negative pole, connected with a large flat electrode, is placed over the abdomen, and a rather strong current, whose power has first been tested by inserting a needle connected with the positive pole in the white of an egg and noting its power to coagulate the albumin, is then allowed to pass for one or several hours. Consolidation of the sac may be very decided immediately after the completion of the operation, but the clots may be subsequently absorbed. There is always the danger of weakening the walls of the sac and producing hæmorrhage, and also a less immediate danger of embolism. Neither of these, however, is sufficient to forbid the operation in suitable cases. The most favorable are saccular aneurisms, or such as have perforated the chest-wall and formed an adventitious sac externally.

The introduction of wire or other filiform material into the sac is a method of treatment instituted by Moore of London, which has sometimes caused obliteration of the aneurism. Of late attention has been particularly directed to

Loreta's method, in which electrolysis is combined with the introduction of wire. Several feet of fine silver wire are passed through a hypodermic needle directly from the spool, so that the wire curls up within. This is attached to the positive pole, the negative being connected with a surface pad or with an insulated needle introduced into the sac. A current is then passed through as in the method of simple galvano-puncture. Good results have been obtained from this method in a number of cases, but final judgment cannot as yet be formed regarding the dangers and the relative value compared with other forms of local treatment. It may be of temporary advantage in some cases to prevent threatened rupture, though no permanent cure be possible. Injection of solutions of perchloride of iron or other styptics is dangerous and valueless.

Many drugs have been advocated for the effect upon the vessels or upon the heart. Many years since Langenbeck advised the injection of ergotine or ergot to contract the sac, but this treatment has properly fallen into disuse. In cases in which temporary over-action of the heart is noted I have sometimes found aconite a valuable remedy. It should be used in doses of from one to two drops every hour or two until the pulse grows more tranquil, if this effect is produced by a few doses; but prolonged use of aconite must be discouraged. When blood-pressure is excessive, nitro-glycerin is a remedy of decided value, but, as a rule, should yield place to venesection. Digitalis has been used to steady the heart's action, but is of more than doubtful value, since it may so increase blood-pressure as to lead to rupture of the sac. In the final stages failing heart-power may render the use of digitalis, strychnine, or alcohol advisable or necessary.

Recent studies of the physiology of coagulation encourage the hope that the use of calcium salts may prove of value in promoting clotting, but practical results have not as yet been obtained.

Certain special symptoms require additional treatment. Pain may be controlled sufficiently by the iodide of potassium, but in the late stages morphine often becomes necessary. Extreme dyspnœa or cyanosis is relieved by free venesection. If moderate and slowly developed, saline purges or elaterium may be of value. Urgent dyspnœa may seem to demand tracheotomy in cases in which spasm of the larynx appears evident, but the operation is generally useless, from the fact that pressure upon the trachea or bronchi is generally present in these instances. Dropsy is treated in a similar manner to that employed in cardiac affections, but active measures must, as a rule, be avoided. Southey's drainage-tubes may be employed and may for a time be of great value. External rupture of aneurisms is sometimes retarded by painting the surface with collodium or solution of gutta-percha, or by applying a shield of metal or felt. The value of galvano-puncture in such cases has been referred to.

Aneurism of the Pulmonary Artery.

Dilatation of the pulmonary artery is not unusual in mitral disease, in phthisis, emphysema, or other conditions which obstruct the lesser circulation.

In some cases the dilatation is very marked, the trunk of the vessel reaching a diameter of as much as two inches, and all of the branches likewise being dilated. Secondary sclerosis and atheroma are not unusual in these cases. Relative insufficiency of the semilunar leaflets occurs when the dilatation becomes marked. There may be visible pulsation to the left of the sternum in the third intercostal space, and not rarely a thrill is felt in addition to the pulsation. On auscultation a systolic murmur, propagated upward and to the left, is generally audible, and if relative insufficiency of the pulmonary valves becomes established, a diastolic murmur may also be heard.

Aneurism of the pulmonary artery is extremely rare, for the arterial changes necessary to the development of aneurism are very unusual in this vessel. Occasionally, however, small saccular or spindle-shaped aneurisms of the trunk, or less commonly of other parts, of the pulmonary artery have been recorded.

The symptoms in case of a large aneurism are practically the same as those which occur in aneurism of the thoracic aorta, but the evidences of disturbance of the pulmonary circulation and of weakness of the right heart are prominent. Cyanosis or lividity, marked dyspnœa, with cough and expectoration, are generally decided symptoms. Pain is rarely absent if the sac has grown to considerable size.

On physical examination pulsation, and even some prominence, may be seen in the third interspace at the left border of the sternum. On palpation the systolic pulsation may be attended by a thrill, and there is sometimes a diastolic shock coincident with closure of the semilunar valve. Percussion over the area of pulsation reveals a dull or flattened note, but the exact extent will depend on the condition of the adjacent lung. Sometimes, indeed, the lung may be so retracted that the percussion-note in the pulmonary region is decidedly dull, though neither dilatation nor aneurism of the artery be present. A loud, superficial systolic murmur is heard on auscultation, and is transmitted to the left and upward. The second sound is distinctly accentuated.

Aneurism of the pulmonary artery rarely reaches large size, but tends to rupture into the pericardial sac. Aneurism and dilatation must be distinguished from the simple pulsation of the pulmonary artery due to retraction of the left lung. The physical signs on palpation and pressure may be the same, but on auscultation the sounds may be entirely normal, or, though a functional systolic murmur be present, the marked accentuation of the second sound is generally absent. When visible bulging is present, the diagnosis of aneurism is almost certain, and especially if pressure-symptoms supervene.

The **prognosis** is highly unfavorable.

Treatment must be conducted on the same principles as obtain in cases of aortic aneurism.

Minute or miliary aneurisms of the terminal branches of the pulmonary artery are not uncommon in pulmonary tuberculosis, and are found most frequently in the branches in the walls of cavities. They result from the localized weakening of the vessel-wall occasioned by degeneration of the adventitia bordering on caseous foci, and frequently rupture, causing hæmoptysis.

ANEURISM OF THE CORONARY ARTERIES.

Aneurism of the coronary arteries is very rare. Any part of either vessel may be affected, and there is generally a high degree of arterio-sclerosis present in the affected portion which has on this account become weakened and dilated. Occasionally embolism leads to dilatation above the point of occlusion, or at that point if the walls of the artery become diseased. The aneurisms are generally quite small, but in marked cases grow to the size of a small nut. There may be one or several on the same vessel.

Age predisposes, because arterial disease is more apt to occur in those of advanced age; but a remarkable case, in which three aneurisms of the coronary arteries were found in a boy of seven years who had died with scarlatinal dropsy, was reported by Dr. Gee. The aneurism may rupture into the pericardial sac or may erode the wall of the heart by pressure, and lead to cardiac rupture. In either case the symptoms are those of rupture of the heart. Before rupture takes place there are no distinctive symptoms, and the condition cannot be recognized during the life of the patient.

The peculiar form of arterial disease described by Kussmal and Maier under the name of "periarteritis nodosa" not infrequently affects the coronary vessels. According to Eppinger, this condition is really one of congenital aneurism.

ANEURISM OF THE ABDOMINAL AORTA AND ITS BRANCHES.

Aneurism of the abdominal aorta is far less frequent than that of the thoracic aorta. It is most frequent in the male sex, between the ages of thirty-five and fifty years, and the causes are the same as in thoracic aneurisms. The usual situation is near the cœliac axis, and frequently the root of this trunk is involved in the sac. Of 103 cases collected by Lebert, but 3 occurred at or near the bifurcation.

Symptomatology.—The most decided symptom and the most constant is pain. At first this may be sharp and shooting in character, radiating from the back, round the abdomen to the umbilicus or down into the legs. Later the pain may become of a dull, gnawing, or boring nature, when erosion of the vertebræ is taking place. Gastric disturbances are frequent, particularly vomiting; and there may be paroxysms of intense gastralgia. Severe colic-like pain may also be due to embolism of the mesenteric artery. Congestion of the kidneys or of the spleen may result from pressure on the corresponding veins. When the bodies of adjacent vertebræ become eroded, paræsthesia of the lower part of the body, and finally paraplegia, may be observed. Jaundice is an occasional symptom.

Physical Signs.—The tumor may be evident on inspection of the abdomen, and may present distinct pulsation. The impulse is felt to be heaving and expansile, and occasionally in aneurisms just beneath the diaphragm a diastolic shock is communicated from the heart. A systolic thrill may be palpable. On auscultation a toneless systolic sound is sometimes heard, but more frequently a soft bruit or murmur. Diastolic murmurs are very rare. The femoral pulse is often retarded and noticeably weak. Frank called attention to the increased tension which may be produced in the femoral arteries by pressure on the aneurism, and to the intermission of a few beats immediately after the pressure is withdrawn. This procedure, however, as well as that of Scheele, who demonstrated the increased tension of the sac when the femoral vessels were compressed, entails grave danger.

Terminations.—Abdominal aneurisms may rupture into the peritoneum, the duodenum, the stomach, the pleuræ, the retroperitoneal space, the bladder, or the spinal canal. External rupture is very rare. Rupture into the vena cava inferior sometimes occurs, and gives rise to great dyspnœa, dropsy, and other evidences of disturbance of the circulation.

Diagnosis.—Aneurisms of the descending part of the thoracic aorta are distinguished by their position and often by evidences of intrathoracic pressure.

Dynamic pulsation of the aorta is a condition in which there is marked undulation, not rarely forcible and visible, and often attended with palpable thrill and with a systolic murmur. It occurs more frequently in emaciated, neurasthenic women than in men. It is to be carefully excluded by the absence of distinct evidences of pressure, and especially by the absence of a palpable tumor. Simple pulsation, thrill, and murmur may be present in either condition.

Severe paroxysms of gastralgia or lumbago of obscure origin should lead to careful physical examination for aneurism.

Prognosis.—The prognosis is extremely grave, though recovery is not impossible. Spontaneous cure is occasionally noted.

The general methods of **treatment** adopted in thoracic aneurism are to be used, and in addition pressure may be made on the proximal portion of the aorta, the patient being kept under chloroform. In one case this treatment was unsuccessful until the third trial was made. Pressure has been continued as long as fifty-two hours. Unnecessary violence must be avoided lest injury be done to the sac.

Aneurisms of the Cœliac Axis are not infrequent in association with aneurism of the upper part of the abdominal aorta, but its branches may also be involved independently.

Aneurism of the Splenic Artery is occasionally met with. Usually the sac is small and there may be several; in rare cases are large splenic aneurisms observed. The symptoms are indefinite in nature. Nearly always there is deep abdominal pain, not rarely referred to the back. A tumor may sometimes be felt, and a systolic bruit is heard on auscultation. Gastric disturbances, vomiting, and even severe hæmatemesis, are sometimes observed. Intraperitoneal rupture may occasion sudden death.

Aneurism of the Hepatic Artery is exceedingly rare, and the symptoms are indefinite in nature. Pressure may be exerted upon the venous trunks or biliary duct, and enlargement of the liver with jaundice may occur.

ANEURISM OF THE SUPERIOR OR INFERIOR MESENTERIC ARTERY is exceedingly rare, but the former vessel is the one most frequently affected. Pain in the epigastric or lumbar region is usually present. A tumor with a systolic murmur may be detected in the middle line of the abdomen, and embolic manifestations may result from detachment of portions of the clots within the aneurism and obstruction of terminal branches of the mesenteric arteries. The termination is usually in rupture into the peritoneal cavity. Compression through the abdominal walls has been practised with the success.

ANEURISM OF THE RENAL ARTERIES is occasionally observed. Small ones are not rare, but large sacs, such as those recorded by Byrom Bramwell, are very uncommon. Rupture into the retro-peritoneal cavity may take place.

STENOSIS OF THE AORTA.

General stenosis of the aorta is a congenital condition occurring most frequently in chlorotic girls, and is associated with imperfect development of the organs of reproduction. Independently, however, of these cases there are occasionally found subjects in whom the aorta remains throughout life of the size it had in childhood. These people are poorly developed, pale, and weak, with reduced vitality and little power of resistance. Virchow called attention particularly to the hypoplasia of the blood-vessels in persons affected with hæmophilia.

When such a condition is present, there is usually also more or less endarteritis, the intima becoming puckered and wrinkled and the seat of fatty degeneration. These conditions may finally lead to the formation of an aneurism or to rupture. Usually the left ventricle is hypertrophied and dilated, and a systolic murmur may be heard over the arch of the aorta.

Localized constriction of the aorta may be found near the ductus arteriosus as a congenital lesion. It leads to dilatation of the vessel on the proximal side, and to various degenerative changes in the walls. On the distal side the vessel may be smaller than normal or may be of the usual calibre. Localized stenosis may also result from pressure of enlarged mediastinal glands or from syphilitic infiltration of the wall of the aorta.

Symptoms may be entirely wanting or there may be evidences of obstruction to the circulation, with consequent hypertrophy of the heart.

RUPTURE OF THE AORTA.

Rupture of the healthy aorta may result from a fall, from severe blows upon the chest, or from direct injuries by stab-wounds or the like. More frequently, however, the walls have been previously weakened by degenerative changes of the media or intima, when sudden strain or cardiac excitement leads to rupture at the point of disease. Aneurism is the most frequent cause of all, but is not, strictly speaking, a cause of rupture of the aorta itself. Very rarely localized or general stenosis of the aorta, especially when cardiac hypertrophy is associated with it, occasions rupture, and instances have been recorded in which the vessel had been involved in the destructive changes of adjacent cancer or ulceration of the œsophagus or of caries of the vertebræ.

The rupture is generally transverse, and is most frequently found just above the aortic valves, the blood finding its way into the pericardial cavity. In other cases the perforation is higher up, and leads into the anterior or posterior mediastinum or into one of the pleural cavities. Rupture of the abdominal aorta is very rare. Rupture of the intima and media may precede that of the adventitia by a considerable interval, and the blood may separate the layers, forming a dissecting aneurism and never leading to complete rupture.

At the moment of rupture there may be severe, agonizing pain, with a sense of laceration or bursting, and with fear of impending death. Later, anæmia, loss of power, and collapse give evidence of the internal hæmorrhage. When the fatal issue is not immediate, physical examination may reveal the existence of liquid in the pleural or pericardial cavities. In cases of rupture of the intima and media, followed after an interval by perforation of the adventitia, a partial recovery from the primary collapse may precede the fatal issue.

The **diagnosis** cannot be made with certainty unless there have been evidences of aortic aneurism.

Treatment, as a rule, is of no avail, but in cases of partial rupture absolute rest, with remedies to quiet the action of the heart and promote general tranquility might insure the formation of a dissecting aneurism and prevent complete rupture.

DISEASES OF VEINS.

PHLEBITIS.

PHLEBITIS, or inflammation of the veins, may be either acute or chronic, the latter resembling in its nature arterio-sclerosis, and therefore receiving the name phlebo-sclerosis.

ACUTE PHLEBITIS.

Acute phlebitis may be of two kinds: thrombo-phlebitis, in which a change takes place in the contained blood before the wall of the vein is affected; and periphlebitis, in which the wall of the vein and the surrounding tissues are first involved. Phlebitis occurs where the circulation is sluggish, and on account of the anatomical conditions is most frequently found in the veins of the lower extremities, in the large veins of the pelvis, or in those of the dura mater.

Etiology and Pathology.—Thrombo-phlebitis is the more common of the two forms, and occurs in conditions in which there is unusually sluggish circulation, and in which there seems to be some alteration in the blood favoring its coagulation. A clot is formed within the vessel, not rarely near one of the valves, and later the vessel-wall becomes altered as in periphlebitis. Thrombophlebitis is common during the puerperal state (phlegmasia alba dolens), and occurs in cardiac affections, in phthisis, after infectious diseases, particularly typhoid fever, in septic conditions, and in other states of lowered vitality.

In periphlebitis the inflammatory changes first affect the vessel-wall, the thrombotic process within being secondary. Nearly always the vessel-wall becomes affected as a result of extension of inflammation from surrounding structures, as in diffuse cellulitis, but the vein may be primarily involved in rare instances, as in cases of chronic phlebitis, in which an intercurrent acute inflammation supervenes, or after traumatism or deligation of the vein.

The outer wall of the vein is principally affected, showing increased vascularity and cellular infiltration, as a result of which the vessel becomes considerably thickened and remains patulous, like an artery, when cut through transversely. The inner coat loses its transparency, becoming of a dull leaden color, and in the later stages may be eroded and stained with blood-pigment from the clot within the vessel. The inflammatory exudate in the outer wall of the vein may form pus, thus giving rise to an abscess in the vessel-wall (suppurative phlebitis), or the whole wall and part of the clot may break down, forming a large abscess. When the thrombus becomes dislodged, it may be carried along in the circulation and cause a sudden fatal termination. On the other hand, if it be rapidly disintegrated, as in suppurative phlebitis, smaller portions are disseminated as emboli and give rise to metastatic abscesses.

After a considerable time the clot may become organized by granulations extending from the vessel-wall, and the vein is thus converted into a fibrous cord—obliterative phlebitis. Calcification may take place in the thrombi in other cases, and leads to the formation of phleboliths or vein-stones. Obliterative phlebitis occurs not only in the small veins, but also in the larger ones, as the femoral, or even the vena cava. Rarely indeed is there complete restoration of the vessel-walls to their normal size. If not obliterated, they are very much thickened and remain so. Suppurative phlebitis, if not fatal, always leads to complete obliteration.

Symptomatology.—The symptoms of phlebitis are those of obstruction, and vary according to the situation and extent of the thrombotic change. Thus a parietal thrombus may cause so little obstruction as to give rise to no symptoms, and a completely obstructing one when situated in vessels with free anastomosis may be similarly free from symptoms. When a large vein is affected, there is usually severe pain with tenderness on pressure. At times the pain is intermittent or neuralgic in character. The limb swells and becomes stiff. If a superficial vein be diseased, there may be soft œdema with distinct pitting on pressure, whereas, if a deep vein be involved, the swelling is tense and hard and more painful. In the latter cases the pain and tumefaction may be the only symptoms, but in superficial phlebitis there is a dusky-red line along the course of the vein, and the vein itself may be felt as a swollen, hard, cord-like structure with knot-like prominences corresponding with the position of the valves.

The temperature of the limb varies. Usually at the outset it is elevated, but later, when the circulation fails and œdema increases, the temperature falls.

The constitutional symptoms depend upon the extent and severity of the inflammation, rarely being grave excepting in suppurative cases, with disintegration of the clot, when pyæmia is apt to be developed. In these cases irregular fever, chills, and sweats give evidence of the toxæmic state.

Diagnosis.—The local conditions require distinction from lymphangitis. The latter almost always depends upon some local injury, is usually more diffuse, presents a brighter redness, and is invariably attended with adenitis of the glands to which the affected vessels lead.

Phlebitis may be suspected in a case of diffuse cellulitis surrounding an important vein if there be a sudden increase of pain and swelling, together with enlargement of the tributary veins.

Deep-seated phlebitis may be mistaken for neuralgia, but the latter is unattended by œdema and marked circulatory disturbances, and is often relieved rather than aggravated by pressure.

Prognosis.—In ordinary cases the prognosis is good, and even where large branches are obstructed by thrombo-phlebitis the collateral circulation is usually established. Suppurative phlebitis, however, is a grave affection, and if it occur in internal organs is usually fatal.

Treatment.—The affected part must be placed as nearly at rest as possible. Undue manipulation or friction of the parts is always attended with the danger of dislodging parts of the thrombus and causing sudden death. The limb should be elevated, and enclosed in cotton-wool retained in place by light turns of a roller bandage. Counter-irritants applied locally are often of great advantage. In mild cases the tincture of iodine may be painted lightly along the course of the vein; in more severe cases blisters may be of value. In cases in which the venous repletion is excessive, leeches applied locally decrease the pain and prevent undue stagnation of the circulation. Warm fomentations may be of value, and occasionally when local inflammatory reaction is marked cold applications are serviceable.

Internally advantage will be derived from the use of quinine and of other remedies directed to the patient's general condition. Opium may be needed when pain is severe.

CHRONIC PHLEBITIS.

Chronic phlebititis or phlebo-sclerosis may result from acute phlebitis or may be a primary, chronic process in veins subjected to increased blood-pressure, which as a result become thickened and varicose. Phlebo-sclerosis may be associated with arterio-sclerosis as a result of the same causes. Among 87 cases of arterio-sclerosis Huchard found sclerosis of the veins in 51.

The walls of the veins become thicker and harder, and more rigid by the slow growth of fibrous tissue, and subsequently hyaline or fatty degeneration or calcification may ensue as in the arteries. In the larger veins atheromatous plates and ulcers are seen, as in the larger arteries. Cases in which the veins as well as arteries are involved have been designated as angio-sclerosis.

Syphilitic phlebitis and periphlebitis are most apt to occur in the ramifications of the portal vein and in the umbilical vein of new-born children.

Chronic phlebitis may occasion more or less impediment to the circulation, but the symptoms are more directly the result of associated arterio-sclerosis.

Rest with counter-irritation is of value in cases in which peripheral veins are involved. Counter-irritation by means of the actual cautery in particular is often attended with happy results.

DILATATION OF THE VEINS.

SYNONYMS.—Phlebectasia; Varix; Varicose veins.

Definition.—Dilatation or varicosity may be met with in any of the veins, but is usually found in those of the lower extremities, in the plexus about the rectum, and in those of the spermatic cord. The superficial veins are more commonly affected, although the deep veins are also more or less involved. Dilatation of the veins is more common in women than in men, probably on account of the obstruction offered to the return circulation from the legs during pregnancy; and it is a disease of later life rather than of young persons.

Etiology.—The dilatation is directly due to some increase of the distending force within the veins. This, as a rule, results from some local obstruction to the venous circulation. At the same time, however, a weakened condition of the walls of the veins themselves plays a part in the etiology, as is evidenced by the fact that phlebectasia is a disease of advancing years and debilitated conditions. Normally, there is greater venous pressure in the veins of the lower extremities than elsewhere, and this excess becomes considerable in old persons with generally sluggish circulation, who by their occupation are required to be on their feet during a large part of the day. The importance of cardiac weakness in the production of dilatation of the veins has too often been overlooked, but is well illustrated in cases occurring in elderly persons with cardiac degeneration or valvular disease. Even though hypertrophy of the heart be present, this may be sufficient only to partially overcome the arterio-sclerosis or valvular defect, and not adequate to maintain the venous circulation as well.

Local obstructions of various kinds may directly occasion increase of pressure within the vein and consequent dilatation. Obstruction to the venous circulation occasioned by cirrhosis of the liver, by the pressure of a gravid uterus or of a distended sigmoid flexure, frequently gives rise to dilatation of the hæmorrhoidal veins. Thrombo-phlebitis or a tight garter may similarly lead to varicosity of the veins of the leg.

Often in mitral disease there is a certain degree of dilatation of the pulmonary veins, and there may be varicosity of the peripheral veins and, in addition, of those of the liver and other viscera, when secondary tricuspid regurgitation supervenes.

Morbid Anatomy.—The veins appear knotted from greater dilatation at one portion than another. The dilatation at one point may affect the entire circumference of the wall, while at another, only a part gives way, leading to a localized bulging or pouch. There is also elongation of the vein, so that it becomes extremely tortuous. If one set of veins be obstructed mechanically, the tributary veins are usually also dilated from the increased work thrown upon them.

Varicosity begins just above the valves, and at this point the whole vein becomes uniformly dilated, so that the valves are rendered incompetent. When the latter condition has become established general dilatation of the veins proceeds more rapidly, for instead of several columns of blood, each supported by a valve, there is now but one pressing backward upon the distal portions of the vein.

The walls are usually thicker than normal, for with the beginning dilatation there is hypertrophy of the muscular coat, and at the same time slow inflammation occurs, with cellular infiltration and later fibrous overgrowth. There is also more or less disturbance of the nutrition of the walls, leading to fatty degeneration and sometimes calcification, although these changes are less common than in the arteries. Rarely the distention is so acute that there is actually thinning of the walls and even rupture of the vessel. When this occurs there is usually a giving way of the overlying skin or mucous membrane and profuse hæmorrhage. With the slowed circulation and inflammation in the walls it is not an uncommon thing late in the disease to find thrombosis within the vessels.

Symptomatology.—The local conditions described above may be plainly visible, or the thickened, tortuous, and knotted veins may be felt beneath the skin. There is generally a dull aching pain in the part, with a sense of fulness or distention, becoming more marked after the patient has been standing upright or walking for some time. In cases in which there is considerable impediment to the circulation there is apt to be œdema of a peculiarly hard and resisting nature, so that the skin does not pit on moderate pressure. Low-grade inflammatory processes are also present, and lead to general induration of the tissues surrounding the diseased veins. Troublesome eczematous conditions of the skin and indolent ulcers result from the tendency to stasis of the blood. In the case of varicose hæmorrhoidal veins considerable local inflammation is frequently met with around the veins.

Treatment.—The first-point in the treatment should always be the removal of the cause when possible. Attention should be paid to the state of the bowels and to the condition of the hepatic circulation. Scrupulous care is required as to every detail of personal hygiene, for the affected veins constitute a point of least resistance upon which the influence of every imprudent exposure or exertion is sure to be expended. In cases in which cardiac weakness is an etiological factor the judicious use of digitalis or nux vomica may be of service, at least for a time.

Locally, elastic bandages or stockings may be applied so as to afford evenly distributed support and pressure. The patient should avoid remaining in the erect posture for long periods at a time.

In cases in which the venous distention and thickening have become fully established surgical treatment may be required.

DISEASES OF THE MEDIASTINUM.

BY WILLIAM PEPPER.

In this section will be considered the affections of the cellular and lymphatic tissues of the mediastinum and the diseases of the thymus gland. The diseases of the pericardium, of the heart, and of the large blood-vessels are included in separate chapters.

MEDIASTINITIS.

Inflammation of the cellular and fibrous tissues of the mediastinum may be acute or chronic. Acute mediastinitis, the commoner and more important condition, runs on to suppuration and the formation of abscess, whereas the chronic form occasions cicatricial adhesions of the pericardial and pleural surfaces and of the other structures in the mediastinum.

ACUTE MEDIASTINITIS, or abscess of the mediastinum, is nearly always traumatic in nature, resulting from penetrating wounds or injury to the sternum, ribs, or other adjacent structures. Occasionally abscess of the mediastinum is idiopathic, arising after exposure to cold; in other cases it follows infectious fevers or pyæmic conditions, and is embolic in nature. The mediastinum may be affected secondarily by extension in caries of the sternum and ribs, in pericarditis, pleurisy, suppurative lymphadenitis, or from rupture of a tuberculous cavity of the lungs.

Males are more frequently affected than females.

Morbid Anatomy.—The purulent collection may occupy the anterior or the posterior mediastinum, but the former is far the more frequent. In its further enlargement it may erode the chest-walls and break externally to the left of the sternum, or it may even perforate the sternum itself. In other cases rupture takes place into the pleural or pericardial sac, into the lungs or bronchi, or into the œsophagus, and rupture of the internal mammary artery, and even of the aorta, has occurred. The abscess may burrow into the abdominal cavity, and may extend as far as the inguinal region.

When the disease remains localized, the pus may become inspissated, and in the case of small abscesses every trace of the disease may become effaced.

Symptomatology.—As a rule, mediastinal abscess is accompanied from the beginning with gradually increasing pain and tenderness under the sternum. Sometimes there is merely a sense of constriction, and pain of a throbbing character is occasionally noted. The patient is restless, loses appetite, and presents irregular fever, and not rarely chills and sweats. The pulse is rapid and weak, and there may be considerable dyspnœa. Decided pressure-symptoms, however, are unusual. Cough is frequent and the expectoration may be blood-tinged. In abscesses of the posterior mediastinum difficulty is experienced in swallowing.

The **physical signs** may be distinctive. Not rarely there is prominence of the upper part of the sternum, and there may be pulsation communicated from the heart. The skin is reddened and may pit on pressure, and on percussion there is dulness under the sternum. The heart-sounds are distant and feeble.

Duration and Prognosis.—The disease may run its course and subside by rupture and discharge of the pus in a few weeks; but sometimes the abscess becomes inspissated and chronic. Grave complications may occur at any time, and the prognosis must therefore be exceedingly guarded.

Diagnosis.—Abscess of the mediastinum is distinguished from aneurism by the absence of expansile pulsation, of diastolic shock, and of the auscultatory phenomena of aneurism. The pressure-symptoms in aneurism are much more marked, whereas irregular fever, chills, and sweats are absent. The history furnishes important points leading to the proper diagnosis.

Tumor of the mediastinum is excluded by the rapid and febrile course of the case, by the acuteness of the pain and other symptoms, and by the absence of secondary deposits in other situations.

The distinction of pericarditis with effusion has been considered in the description of that disease.

Treatment.—The patient must be kept at rest and given nutritious though easily assimilable diet. In the early stage ice-bags or Leiter's coils applied over the sternum give great relief and probably limit the severity of the inflammatory process. Leeches are also of value, and may always be used when the indications of vascular engorgement are present.

In a number of instances the sternum has been trephined and drainage established with happy result; but, as a rule, it may be well to wait for some evidence of pointing. If fluctuation is present, the abscess should always be opened.

In the later stages stimulating and supporting remedies will be required, and at any time signs of cardiac weakness demand the use of alcohol, ammonia, or other stimulus.

CHRONIC MEDIASTINITIS, or mediastino-pericarditis, has been considered in the description of Pericarditis. Occasionally abscess of the cellular tissue of the mediastinum becomes chronic, or tuberculous lymphadenitis may occasion chronic purulent collections. The latter will be included in the description of Tuberculous Lymphadenitis.

ACUTE LYMPHADENITIS.

Etiology.—The lymphatic glands surrounding the bronchi and lying upon the spinal column in the posterior mediastinum are enlarged and inflamed in a variety of affections of the lungs, principally in the bronchitis of influenza, measles, and whooping cough, and in broncho-pneumonia. Mediastinal lymphadenitis may also occur in more remote infectious processes, such as typhoid fever, facial erysipelas, and diphtheria. Peter and de Mussy held that nasopharyngeal diseases frequently occasion enlargement of the tracheal and bronchial glands. Acute lymphadenitis is more frequent in young persons than in those of maturer age.

Morbid Anatomy.—The glands become enlarged, and are soft and moist from the presence of inflammatory œdema. Their color is usually light pink or brownish, but in the later stages turns to a grayish hue. If the inflammatory process be intense, abscess may result, but this is less frequent than in tuberculous lymphadenitis. If the inflammation be long continued, permanent enlargement with sclerosis of the glands may follow.

Symptomatology.—In exceptional cases the glandular enlargement may be sufficient to give rise to serious pressure upon important structures, and the symptoms then are those met with in mediastinal tumor. As a rule, however, the glands are only moderately enlarged, and the symptoms are due to the surrounding congestion with irritation of the adjacent nerves. The clinical history is very like that of whooping cough during the paroxysmal stage; indeed, the similarity is so great that de Mussy regards lymphadenitis as a constant condition in whooping cough and as the cause of the paroxysms.

The most marked symptom is the peculiar paroxysmal cough, which may be followed by vomiting, but rarely terminates with the shrill inspiratory effort so common in whooping cough. Paroxysms of dyspnœa are also met with, and there may be disturbance of the circulation, leading to cyanosis when the child exerts itself in play or otherwise. Hoarseness is not unusual.

Physical examination may disclose a moderate degree of dulness under the upper part of the sternum or posteriorly between the scapulæ; but more frequently percussion discovers no abnormal conditions. On auscultation sonorous and sibilant râles are frequent; and the breath-sounds may be noticeably weaker on one side in cases where pressure is exerted upon one or the other bronchus.

Diagnosis.—It is most important to distinguish simple bronchitis with lymphadenitis from whooping cough; but in many cases this diagnosis can only be made after the most searching examination. Evident communication of the disease by contagion, and a regular course consisting of a primary febrile stage, a stage of paroxysms, and a final stage of simple bronchial catarrh, would point strongly to whooping cough. The paroxysms themselves are almost identical in the two affections, though it has been noted that the shrill inspiration following the seizure is more frequently absent in lymphadenitis. Extreme dyspnœa, coming on in paroxysms, and diminution in the breath-sounds and tactile fremitus, would also point to glandular enlargement.

Prognosis.—With the subsidence of the primary bronchitis the glands usually diminish in size and the urgent symptoms are relieved. In some cases, however, chronic hypertrophy of the glands may follow, and may lead to constant dyspnœa, especially when the naso-pharyngeal lymphatic tissues are coincidently hypertrophied. Abscess of the mediastinum may result from suppurative inflammation of the glands, and may terminate fatally. According to Baréty, the pressure of the enlarged glands upon the pulmonary artery predisposes to pulmonary tuberculosis.

Treatment.—It is of first importance to treat the bronchitis upon which the lymphadenitis depends. Rest in bed and nourishing diet must be insisted upon. Attention to every detail of personal hygiene is demanded. Counter-irritation by tincture of iodine may be used posteriorly between the scapulæ or anteriorly over the upper part of the sternum. Iodide of potash and arsenic are regarded with reason as favoring a reduction in the size of the glands. Mercurial inunctions are especially to be recommended. Quinine may have some influence in controlling inflammation.

TUBERCULOSIS OF THE MEDIASTINAL LYMPH-GLANDS.

The lymphatic glands situated at the roots of the lungs are regularly enlarged in pulmonary tuberculosis, and may grow to considerable size. There is, however, a group of cases in which the lungs remain healthy, while the mediastinal glands are decidedly involved. In cases of this kind the infection takes place by a penetration of the bacilli through the lining membrane of the air-passages without effecting any local change. In dealing with cases of suddenly-disseminated tuberculosis when the primary seat of disease is obscure, the lymphatic glands of the mediastinum must always be examined. The disease is much more frequent in children than in persons of greater age.

Morbid Anatomy.—The glands are at first swollen, red, and soft, as in simple lymphadenitis. On close inspection tubercles may be discovered in the substance of the gland, but very soon there is cheesy necrosis affecting part, and finally the whole, of the gland. Neighboring glands tend to fuse into a single mass, which may be surrounded by a more or less resisting connective-tissue membrane. Sometimes the glands break down completely, and a cyst containing puriform liquid results. If the glandular mass be large, injurious pressure may be exercised upon the bronchi, nerves, or blood-vessels near by. A more serious accident is rupture into the œsophagus, trachea, bronchi, or pulmonary artery. Traction diverticula usually precede the rupture in these cases. The lungs, the pleuræ, or the pericardium may be secondarily involved by extension or by rupture of the glands.

Symptomatology.—The nature of the symptoms will depend upon the manner of involvement of the mediastinal glands, whether primarily or secondarily after pulmonary tuberculosis. In the latter case the patient will present the symptoms of phthisis, with cough, expectoration, fever, and emaciation; but in proportion to the degree of glandular enlargement the cough becomes more and more paroxysmal, and may finally be quite indistinguishable from that of whooping cough. In the intervals between these paroxysms, however, there may be the ordinary cough of phthisis. When the glands are primarily involved or when the pulmonary involvement is slight or latent, the symptoms are the same as those seen in simple lymphadenitis—paroxysmal cough, dyspnœa sometimes simulating asthma, hoarseness; later, cyanosis, dilatation of the veins, and œdema. Very considerable enlargement of the glands may cause a large tumor, which would occasion the same pressure-symptoms and physical signs as other growths.

Diagnosis.—The supervention of glandular involvement in a case of pulmonary tuberculosis is plainly indicated by the paroxysmal character of the cough, by the increase of dyspnœa, and, on physical examination, by the decrease of vocal and tactile fremitus and of the breath-sounds on one or the other side. There may also be dulness on percussion posteriorly between the scapulæ, but this is frequently wanting.

From simple lymphadenitis, glandular tuberculosis would be distinguished by the greater constitutional depression of the patient and by the more marked evidences of pressure. The distinction from whooping cough is made in the same way as in the case of simple lymphadenitis.

Prognosis.—Doubtless some cases in which circumscribed foci of tuberculous disease have been present in the lymph-glands recover entirely, and calcification of the gland prevents further spread; in other cases a fibrous capsule may limit the spread of the disease for a time; but, as a rule, the neighboring structures become involved, or disseminated tuberculosis results from entrance of the bacilli into the blood. Death may result from perforation of the œsophagus, bronchi, or trachea, or from erosion and rupture of the pulmonary artery or aorta.

Treatment.—Careful nursing, with change of climate, and suitable diet are the most important elements in the treatment of the disease. Cod-liver oil, iron, and iodine are remedies of decided value. The syrup of the iodide of iron may be given in full doses three times daily, or the remedies may be used separately in some other form. Local counter-irritation may be used as in simple lymphadenitis, but is less valuable here.

MEDIASTINAL EMPHYSEMA.

Emphysema of the cellular tissues of the mediastinum sometimes follows the operation of tracheotomy. It may also result from rupture of peripheral air-vesicles in the expulsive stage of labor or in whooping cough or croup, or from perforation of ulcers in the trachea, bronchi, œsophagus, and even the stomach. The air may gradually extend to the tissues of the neck and over the entire surface of the body, or it may be confined to the mediastinum.

The symptoms are not distinctive, and sometimes are entirely wanting. Usually there is more or less unrest or vague fear, with a sense of constriction at the heart. There may be in addition considerable dyspnœa, with alteration in the voice, and sometimes even difficulty in swallowing.

On physical examination the cardiac dulness and the apex-beat are absent, and on auscultation various rustling or crackling sounds may be audible.

Mediastinal emphysema is readily distinguished from pneumopericardium or pneumothorax when the history, symptoms, and physical signs are carefully considered.

The prognosis is often grave on account of the underlying conditions which occasion the emphysema, but the air may be reabsorbed and recovery ensue.

The treatment must be entirely symptomatic. Anodynes and quieting remedies will usually be required, and the strength of the patient will call for stimulants.

MEDIASTINAL HÆMATOMA.

Small hæmorrhages occur in the mediastinal tissues in severe cases of jaundice, hæmophilia, and in other conditions where there is great tendency to extravasation of blood. Larger hæmorrhages result from erosion of blood-vessels, as in abscess of the mediastinum and various destructive lesions of the sternum or ribs, or from traumatic rupture of a vessel. Rupture of an aneurism may occasion a sudden and extensive hæmatoma.

If the hæmorrhage be large, the patient rapidly sinks into a condition bordering on collapse, as in other internal hæmorrhages, and there may at the same time be evidences of pressure upon the mediastinal structures. According to Daudé, ecchymoses make their appearance in the lumbar region after two or three days. Sometimes hæmatoma leads to abscess of the mediastinum.

Physical examination would reveal a considerable increase of dulness in the region of the heart, with disappearance of the apex-beat and muffling of the heart-sounds, in case of a large hæmatoma in the anterior mediastinum. Pericardial effusion would be excluded by the rapid onset, the symptoms of internal hæmorrhage, and the absence of friction-sound.

The patient must be kept absolutely at rest, and should be tranquillized by small doses of morphine. Ice should be placed over the præcordia. If signs of cardiac weakness are distinct, gentle stimulation may be permitted, but active stimulation should not be used.

TUMORS OF THE MEDIASTINUM.

Mediastinal tumors may take origin in the cellular tissue or lymphatic glands, in the thymus gland, or in the pleuræ, pericardium, or bones surrounding the mediastinum. In rare cases a new-growth of the thyroid gland extends in a downward direction and occupies the mediastinum. Among the tumors which have been observed are sarcoma, carcinoma, lipoma, fibroma, osteoma, dermoid and hydatid cysts. Simple cysts have also been met with, and Virchow described under the title "Teratoma Myomatodes" a form containing striped muscle-fibres.

It is difficult to estimate the relative frequency of sarcoma and carcinoma, the two commonest forms, on account of the confusion in nomenclature and the uncertainty as to the nature of lymphadenoma, lymphomata, and other glandular enlargements. If these are considered as sarcomata, this form is the most frequent, but if a separate group of lymphomata is retained, carcinoma is the commoner tumor. In Hare's table of 520 cases, 134 were cancer, 98 sarcoma, and 21 lymphoma. Undoubtedly in the older reports sarcoma was often called cancer. Practically and pathologically, however, it is difficult to distinguish lymphoma from sarcoma.

Mediastinal tumors are more common in the male than in the female sex, the proportion, according to Eichhorst, being three to one. The age of the patient is usually between twenty and forty. Cancer occurs somewhat later than sarcoma, but earlier here than in other situations.

Sarcoma and lymphadenoma of the mediastinum arise in the majority of cases from the lymphatic glands surrounding the bronchi or from the cellular tissue of the anterior mediastinum, and are frequently primary. The growths are irregularly nodular, of grayish color, and usually quite moist. Neighboring structures are involved by direct extension, and distant organs by metastasis. The growth in the majority of cases presents the microscopic features of lympho-sarcoma.

Carcinoma of the mediastinum may be primary or secondary. The former takes origin from the remnant of the thymus gland or from the thyroid; the latter, which is much more frequent than secondary sarcoma, springs from the lymphatic glands or may invade the mediastinum by the direct extension of œsophageal or gastric cancer. The growth is generally soft and juicy, and in the majority of cases belongs to the medullary variety. Cases of epithelioma have been reported by Horstmann and Letulle.

Lipomata and fibromata arise in the fatty and cellular tissue, but are exceedingly rare. Osteomata appear in the form of osteophytes springing from the sternum or, more rarely, the vertebræ in scrofulous or syphilitic individuals.

Dermoid and hydatid cysts are exceedingly uncommon. The former, of which Marfan collected twelve instances in addition to his own case, may reach the size of a fœtal head and present the characteristics of dermoids elsewhere. The usual seat is the region of the thymus gland, in which they are supposed to originate. But 8 cases of hydatid cysts are tabulated in the 520 collected cases of Hare's monograph.

Clinically, aneurism might be regarded as a form of mediastinal tumor, but it has been treated of elsewhere. Tuberculous disease of the mediastinal glands may likewise occasion enlargement sufficient to render the case in its clinical features practically identical with mediastinal tumor; and the same may be said of syphilitic gummata, but these are exceedingly rare.

Symptomatology.—The symptoms of mediastinal tumor are principally the result of pressure upon the surrounding structures. One of the earliest, and generally the most distinctive, is dyspnœa. This may result from pressure upon the lungs, the trachea, bronchi, or upon the vagus. At first there is merely slight shortness of breath, but with the increasing size of the tumor the most pronounced grade of orthopnœa may be developed. It may be noted in some cases that in certain positions the patient's breathing is greatly relieved, and he may habitually assume such a favorable posture. In cases in which the growth presses upon the trachea or bronchi intense dyspnœa is frequently noted, and there may finally be suffocation from extension into the lumen of the air-passages themselves. Paroxysmal dyspnœa, resembling asthma, is met with when the vagus is compressed.

More or less cough accompanies the difficulty in breathing, and profuse expectoration is not infrequent. The sputa may be muco-purulent or more rarely hæmorrhagic, and in cases of dermoid and hydatid cysts hairs and hydatid vesicles, respectively, have been expectorated after violent paroxysms of coughing. Occasionally the cough is very like that of whooping cough, and when the recurrent laryngeal nerve is compressed the peculiar brazen cough of aneurism is observed. In the latter cases also there is usually more or less alteration in the voice.

Injurious pressure may be exercised upon the superior vena cava, with the result that the circulation in the face, neck, and arms is more or less impeded. In mild cases there is noted blueness in the lips and fingers or fulness of the veins of the neck or upper thoracic region. In severe cases great repletion of the veins may be found, and large anastomoses are established between the upper thoracic and lower thoracic and abdominal veins, the blood finding its way into the heart through the inferior vena cava. There may even be extreme œdema of the arms and head.

More frequently there is congestion of the lungs from compression of the pulmonary vein, and in severe cases there may be hæmoptysis.

Compression of the arteries is much less frequent than that of the veins, but a number of instances have been recorded in which there was decided constriction of the aorta. Sometimes, as in the case of Witthauer, a mass of enlarged glands surrounds the aorta and causes an annular stenosis similar in its effects to narrowing of the orifice by rigid valves.

Pain in the chest is frequently noted, and may become the most urgent symptom, but it is remarkable how large a mediastinal growth may develop without causing any pain whatever. In a recent case under my own observation there was a large sarcomatous growth, which had increased very rapidly and which caused decided bulging of the upper part of the sternum, and yet the patient never suffered pain. Among the cases collected and analyzed by Bramwell it was noticeable that pain was much more frequent and persistent in aneurism than in solid tumor.

Pressure upon the œsophagus occurs more especially in tumors of the posterior mediastinum, and gives rise to dysphagia and consequent emaciation. Pressure upon the thoracic duct may occasion rapid and extreme emaciation.

In addition to these symptoms there may be indications of complicating pleurisy, pericarditis, or various pathological conditions of the lungs.

The general condition of the patient depends to a large extent upon the nature of the growth and upon its situation. In cases of carcinoma wasting and cachexia are usually early manifestations, whereas in sarcoma these symptoms are gradually developed, and in benign tumors not until the growth interferes with swallowing or other organic functions. Examination of the blood reveals a more or less pronounced grade of anæmia, especially in cancer; and in cases of mediastinal sarcoma leucocytosis is not infrequent. In some of the latter, indeed, the disease may present the clinical features of leukæmia, as in the interesting case of Palma recently reported.

Physical Signs.—On inspection cyanosis or œdema of the upper part of the body may be a striking feature, and the patient's breathing may be decidedly labored. The enlarged thoracic and abdominal veins may stand out prominently as tortuous cords, and the veins of the neck are greatly distended. Not infrequently the upper part of the sternum bulges forward, and the inner end of the clavicle may be dislocated. Sometimes there is complete perforation, by erosion, of the sternum, and frequently the tumor pulsates visibly. The impulse, however, is rarely as decided as in aneurism, and the diastolic shock is not observed. Enlargement of the cervical lymphatic glands may be noted, and metastatic deposits may occur in other parts of the body. On percussion there is dulness over the prominence, or, in the absence of prominence, over the upper part of the sternum, when the tumor occupies the anterior mediastinum. In those situated in the posterior mediastinum dulness may be found between the scapula and spine on one or the other side. There is also frequently some loss of voice on percussion of the lungs, due to compression or collapse, and on auscultation the breath-sounds and vocal resonance may be decidedly weaker on one side than on the other. A valuable sign indicative of obstruction to one bronchus is the early diminution of tactile fremitus on the affected side. The heart, spleen, and the left lobe of the liver may be decidedly displaced from their normal positions.

Diagnosis.—Tumor of the mediastinum must first of all be distinguished from aneurism of the thoracic aorta, and in most cases the diagnosis is easy. There are instances, however, in which marked evidences of intrathoracic pressure are present, but in which the nature of the growth, whether solid or aneurismal, is extremely difficult to determine. The points favoring aneurism are—sharp systolic impulse with diastolic shock, distinct bruit and accentuation of the second sound, early appearance of pain which radiates to the shoulders and arms, and alterations of the pulses and pupils. Tracheal tugging may be present in cases of tumor, but much less commonly. Decided cachexia, leucocytosis, and the discovery of secondary nodules in the cervical glands or elsewhere, on the other hand, would make the diagnosis of a neoplasm almost positive. The distinctions of mediastinal abscess and of pericardial effusion from tumor have been considered in the description of the former affections.

The differentiation of the various forms of mediastinal tumors depends upon the history and the general condition of the patient. Benign tumors are so rare that they need rarely enter the discussion. In the cases in which hydatid pearls have been expectorated, or in which hairs appeared in the sputa, the diagnosis of echinococcus cyst and of dermoid cysts, respectively, was readily arrived at. Cancer, as a rule, appears later in life, is less rapid in growth, but affects the general nutrition more than sarcoma or lymphadenoma. The latter, on the other hand, more frequently occasion pressure upon the veins and bronchi, and may be attended by marked leucocytosis.

Prognosis.—The prognosis is always extremely grave. The average duration of life is from three to seven months, but cases have been recorded in which the fatal issue was delayed as much as six years.

Treatment.—Surgical treatment alone can avail to effect complete recovery, and has been successfully applied in cases of osteoma of the sternum, and even in a case of sarcoma of the anterior mediastinum. As a rule, however, the nature of the growth and the situation prevent complete removal.

Palliative remedies will be required to quiet the pain or to obviate dyspnœa. In the late stages opium must always be employed to promote euthanasia. Complications, such as pleural or pericardial effusion or pneumonia, must be dealt with according to the exact conditions present.

DISEASES OF THE THYMUS GLAND.

Hypertrophy of the thymus gland is occasionally met with in autopsies on children, especially in cases of sudden death. In a case of this kind in my own experience the gland was greatly enlarged, extending downward and obscuring a large part of the heart from view. The child had died suddenly, and no other pathological condition was discovered. Hypertrophy of the thymus is also regarded as the cause, in some cases, of the spasmodic laryngeal affection known as thymic asthma.

Abscess of the thymus gland has been found in cases of congenital syphilis and occasionally in non-syphilitic children.

Hæmorrhage into the thymus gland may occur in any of the hæmorrhagic diseases, being a frequent lesion in fatal cases of purpura and scurvy.

Sarcoma and carcinoma may both originate in the thymus, but are indistinguishable in their clinical features from malignant tumors springing from other parts of the anterior mediastinum. In leukæmia and Hodgkin's disease the thymus is frequently the seat of secondary nodules, and, as in the case of Palma, may be the original seat of disease.

DISEASES OF THE NOSE.

BY JAMES C. WILSON.

ACUTE NASAL CATARRH.

Definition.—Acute catarrhal inflammation of the mucous membrane of the nasal cavities.

SYNONYMS.—Coryza; Acute rhinitis; Cold or Cold in the head.

Etiology.—Acute nasal catarrh is very common. It is in most instances an independent affection, but it also frequently occurs in the course of the acute infectious diseases. Occurring as an independent affection, it often follows exposure to cold or damp, especially when such exposure is partial, as in wetting the feet or sitting upon damp ground, and conjoined with fatigue or overheating. It is likely to prevail extensively in cold, damp, and changeable weather, when it not rarely assumes the form of local epidemics. These outbreaks are to be distinguished from epidemics of true influenza or grippe, to which they bear a superficial resemblance. House-epidemics of coryza occasionally arise under circumstances that point to the contagiousness of the affection. It certainly is transmissible by means of the handkerchief. In view of the well-known power of the nasal mucous membrane to resist ordinary irritants, this fact has led to the assumption that the affection under consideration is due to a micro-organism. Children are especially prone to it, the attacks promptly following any improper exposure to cold or wet. It may also arise in infants in consequence of gastric or intestinal irritation, indigestion, or the presence of intestinal worms, and is not infrequent as the result of traumatism inflicted by foreign bodies—buttons, grains of corn, pebbles, peas, cherry-pits, and similar objects—which are introduced into the anterior nasal chambers and overlooked.

Transient coryza often results from the action of mechanical or chemical irritants upon the nasal mucous membrane. Among these are dust, smoke, ipecacuanha, and the fumes of ammonia, bromine, and iodine. The troublesome coryza which in many cases follows the internal administration of iodine must, like that caused by the presence of ipecacuanha in even minute amounts, be attributed to idiosyncrasy.

Coryza occurring as a manifestation of acute constitutional infection is an early and prominent symptom of measles, influenza, and pertussis. It is sometimes associated with the ophthalmia of the new-born as the result of infection, frequently gonorrhœal, incurred during parturition, and occurs as an early manifestation of congenital syphilis.

Symptomatology.—Acute nasal catarrh occurring as an independent affection is attended by symptoms of constitutional disturbance. The attack begins suddenly, with chilliness or shivering, a decided feeling of malaise, headache, and repeated sneezing. There is feverishness rather than fever, though exceptionally the temperature rises to 100°–101° F., with slight quickening of the pulse, a dry skin, and muscular pains. The subjective sensations of discomfort are altogether out of proportion to the actual morbid phenomena, which are not usually of great intensity. At first, owing to the inflammation of the nasal mucous membrane and the arrest of its normal secretion, and to the turgescence of its underlying erectile tissue, the nose feels unpleasantly dry and stuffy, and mouth-breathing is necessary. The sense of smell is gone, that of taste greatly impaired; the voice acquires a peculiar, so-called nasal, twang; and nursing infants, being unable to breathe through the nose, are suckled with difficulty. The catarrhal inflammation tends to invade the contiguous mucous tracts. Hence swelling of the tear-ducts leads to lachrymation, and there is frequently an associated conjunctivitis of mild type; the pharynx is red and puffy, and swallowing is attended with pain. If the Eustachian tubes are involved, hearing is impaired, and the extension of the process downward frequently gives rise to laryngitis and bronchitis. Invasion of the sinuses communicating with the nasal chambers occasionally takes place, and gives rise, especially when there is accumulation of secretion, to distressing symptoms, as frontal and facial pains, increased fever, sleeplessness, and complete inability to take food. In the course of a few hours from the beginning of the attack there is established a flow of thin, clear, irritating secretion, which often excoriates the edges of the nostrils and the upper lip, and renders the use of the handkerchief painful. This discharge sometimes causes an eczema of the lip, which may form the starting-point of an attack of facial erysipelas. Not infrequently there is an intercurrent attack of herpes labialis. In the course of the second or third day the secretion becomes muco-purulent. It is now opaque, thick, tenacious, and abundant, and tends to accumulate in the nasal cavities. The swelling of the mucous membrane subsides, nose-breathing is resumed, and with a gradually diminishing secretion complete recovery takes place within a week or ten days. Repeated attacks of the acute affection tend to produce, in persons who are predisposed to catarrhal inflammations, the chronic form of the disease. A large proportion of the cases are subacute. The symptoms are local and of moderate intensity, there is little or no constitutional disturbance, and the attack runs its course in two or three days.

Diagnosis.—There is, under ordinary circumstances, no difficulty in the diagnosis of simple acute nasal catarrh. Healthy new-born infants are not likely to suffer from snuffles. This affection, associated with ophthalmia, is probably in all instances due to the same specific infection. When due to syphilis, it is associated with characteristic lesions of the skin and of the mucous surfaces elsewhere, with notable malnutrition, and it is persistent. Acute nasal catarrh in children, due to the lodgment of foreign bodies, is unduly prolonged, and the discharges are, after a time, likely to be admixed with blood. Furthermore, it is almost always one-sided. In such cases a careful examination of the nasal chambers must be made. It is important to avoid the error of treating for acute coryza a patient who has been taking iodine or one of its preparations without first withholding the drug. The progress of a case of measles or influenza will speedily dissipate any uncertainty as to the nature of the acute catarrh with which each of these diseases begins.

Treatment.—In the milder cases little treatment is required. The patient, though inconvenienced, is able to go about and attend to his affairs. A hot foot-bath at night, with a Dover's powder, followed in the morning by a single dose of quinine, gr. viij–xij, very often appears to exert a favorable influence upon the severity and duration of the attack. Repeated doses of quinine and very large doses are useless and add to the patient's discomfort. A Turkish bath sometimes seems to break up a cold. When the constitutional disturbance is marked, and especially when fever is present, the patient should keep his room, or even his bed, for a day or two. Under such circumstances repeated, small doses of Dover's powder, gr. ij–iij, every two or three hours, or Tully's powder (pulvis morphinæ compositus) in corresponding doses, will prove of service. The value of opiates administered in this manner appears to have been overlooked in the treatment of the severe cases of acute coryza.

In the early stage nothing is so efficacious in relieving the symptoms caused by the swelling of the mucous membrane as cocaine hydrochlorate. It may be used in from 2 to 4 per cent. solution as a spray or snuffed into the nostrils, or as a snuff, 2 grains to a drachm of starch or powdered acacia. Free inunctions of the brow and nose are useful, especially in children, and the edges of the nostrils and upper lip may be protected from excoriation by simple ointments or cosmolin. As the swelling of the mucous membrane begins to subside, and the secretion becomes more abundant, the following powder may be advantageously used as a snuff:

℞. Morphinæ hydrochloratis,	gr. ij;
Bismuthi subnitratis,	ʒij;
Pulvis acaciæ,	ʒiss.—M.

Seiler has devised a soothing and detergent solution which gives great relief used as a spray or snuffed into the nostrils at intervals, especially when the secretion tends to be become inspissated and form crusts. The following is a modification of his formula: ℞. Sodii bicarbonatis, Sodii biboratis, *aa.* ʒss; Sodii benzoatis, Sodii salicylatis, *aa.* gr. ij; Sodii chloridi, gr. vij; Eucalyptol, Thymol, *aa.* gr. j; Menthol, gr. ss; Olei gaultheriæ, gtt. j; Glycerini, f℥ss; Alcoholis, fʒj; Aquæ, q. s. ad Oj.—M. In children this solution may be applied by means of a piece of cotton. A somewhat similar solution may be made by dissolving in two ounces of hot water one of the compound thymol tablets (Seiler) sold in the shops. Nasal lotions must be warmed before being used. As a rule, astringent powders and lotions are unnecessary, often harmful, and should not be employed. If the secretion continues to be troublesome, a few applications of methyl-violet (blue pyoktanin) by means of a cotton-carrier will usually bring it to an end.

CHRONIC NASAL CATARRH.

Definition.—Chronic catarrhal inflammation of the mucous membrane of the nasal cavities. According to the lesions produced, the cases may, in a general way, be arranged into three groups—rhinitis simplex, rhinitis hypertrophica, and rhinitis atrophica.

Etiology.—Repeated attacks of acute nasal catarrh may finally end in the chronic form of the disease. Undue habitual exposure to cold and draughts, to a changeable and humid atmosphere, and the constant inhalation of dust are among the causes of chronic rhinitis. The development of the disease is favored by insufficient food, inadequate clothing, improper ventilation, want of sunlight and fresh air, and other unhygienic conditions. Cachectic states predispose to chronic nasal catarrh, and it is frequently a manifestation of local syphilitic or tuberculous processes. Finally, the protracted nasal catarrh and hypertrophic nasal catarrh of early life tend, in the course of time, to assume the atrophic form—a fact which emphasizes the importance of the prompt and efficient treatment of every case of rhinitis.

Among the common local causes of chronic nasal catarrh are the following: Congenital asymmetry of the nasal fossæ, with marked deflection of the septum; hypertrophy of the adenoid tissue in the vault of the pharynx; traumatism; foreign bodies; and nasal polypi.

Simple Chronic Nasal Catarrh (Rhinitis Simplex).

This term may be used to designate the transitional condition between prolonged or neglected acute catarrh and that in which hypertrophic or atrophic lesions are present. It is also applicable to the chronic catarrhal inflammation due to causes other than acute rhinitis, which has not yet produced such lesions. The mucous membrane is irritable, and there is a constant sensation of discomfort in the nose. Catarrhal disturbances follow every trifling exposure. The erectile tissue is relaxed, and is readily distended with blood, so that one or both nostrils are frequently occluded. The secretion is increased; it is variable in consistency, being sometimes thin and watery, sometimes thick and tenacious. Upon rhinoscopic inspection the entire mucous lining of the nasal chambers is seen to be red, watery, and irregularly swollen.

Chronic Hypertrophic Nasal Catarrh (Rhinitis Hypertrophica).

The symptom-complex comprises interference with nasal respiration, constant, often abundant, discharge of mucus or muco-pus, frequent sneezing, nasal cough, hawking and expectoration of tenacious mucus, dryness of the throat, habitual mouth-breathing, especially at night, with disturbed sleep. The voice acquires a peculiar nasal quality, and the hearing is very frequently impaired. In infants the inability to take nourishment without constant and prolonged interruption for respiration leads to malnutrition, and the nasal obstruction sometimes causes attacks of suffocative spasm. In older children habitual mouth-breathing, with its resulting disturbances of function, begets a peculiar, dull facial expression, mental hebetude, and retardation of the development of the thorax, with characteristic deformities. The mucous membrane of the nasal chambers is congested throughout, and its epithelial and subepithelial tissues are hypertrophied. The constant and characteristic lesion is found in permanent and decided enlargement of the turbinated bodies. The changes in these structures consist of marked increase in the connective tissue, with cell-infiltration, great dilatation of the sinuses of the erectile tissue, and loss of contractibility in their walls. In a large proportion of the cases, especially in the young, there is hypertrophy of the adenoid tissue in the vault of the pharynx, and catarrhal or follicular pharyngitis—an association that has given rise to the term "naso-pharyngeal catarrh."

CHRONIC ATROPHIC OR DRY NASAL CATARRH (RHINITIS ATROPHICA, RHINITIS FŒTIDUS ATROPHICUS, OZÆNA).

A chronic affection of the nose, constituting the terminal stage of neglected cases of rhinitis simplex and rhinitis hypertrophica. It is characterized by atrophy of the mucous membrane, with shrinkage of the turbinated bodies and diminution of the nasal secretion, which becomes muco-purulent or purulent and undergoes inspissation, with the formation of adherent and offensive crusts. Upon rhinoscopic inspection grayish crusts are seen, the removal of which exposes a smooth, pale, or, more commonly, an irritable, slightly excoriated, mucous surface. Actual ulceration is rarely encountered. The turbinated bodies are greatly reduced in size, owing to obliteration of their cavernous sinuses and contraction of their connective tissue. The entire lining membrane of the nostrils is atrophied. The mucous membrane of the pharynx is often in a like condition, being dry and glazed. The sense of smell is lost. Ozæna is present in a large proportion of the cases, but not in all. When present it constitutes a peculiar penetrating and almost insupportable stench. It cannot be regarded as an absolutely diagnostic symptom, as odors having the same intensity and peculiarities are occasionally encountered in other affections of the nose attended with ulceration, as syphilis, the traumatism produced by foreign bodies, and caries and necrosis due to other causes. Whether the stench called ozæna is to be attributed to especially favorable conditions for the development of putrefactive germs in the nose or to some special organism has not yet been determined.

Atrophic rhinitis is more common in females than in males. It is likely to supervene in neglected cases of chronic moist or hypertrophic nasal catarrh before or about the age of puberty.

The prognosis in confirmed cases is, as regards cure, hopeless; as regards relief from the formation and retention of crusts and from the odor, much may be accomplished, so long as a judicious treatment is persistently followed out.

Treatment.—The prophylaxis of chronic nasal catarrh consists in the avoidance of those conditions which produce the acute disease, and in the prompt and judicious treatment of the acute cases, continued until convalescence is established. Especially must the treatment be thorough when the patient manifests a peculiar disposition to catarrhal affections and when congenital nasal defects exist. Abnormalities should so soon as discovered be referred to the specialist for treatment and correction. Systematic treatment of rhinitis must be instituted from the beginning of the disease, whether it occur in infancy or childhood or later in life. This fact cannot be too strongly urged upon the practitioner. To neglect the nasal catarrhs of childhood is to permit to escape the period when they are most amenable to treatment, and too often to doom the patient to a lifetime of discomfort and wretchedness.

The management of rhinitis simplex consists in the frequent removal of the secretions by means of detergent and antiseptic washes applied by means of atomizers or post-nasal and anterior nasal syringes. For this purpose lotions of sodium bicarbonate, biborate, or benzoate may be employed in the strength of from 2 to 5 grains to the ounce of water, to which about seven-tenths of 1 per cent. of sodium chloride have been added, together with a small amount of glycerin and some antiseptic. Seiler's lotion, the formula for which is given on a preceding page, is an excellent one. Astringents should be rarely used, and always in very weak solution. Unguents exert a favorable influence upon the inflamed mucous membrane, and tend to prevent the formation of crusts. They should be bland and unirritating, and may be sprayed into the nostrils or applied by means of a brush or cotton-carrier. Among the most useful and pleasant of this group of intranasal medicaments is warmed vaselin in the form of a fine spray.

In hypertrophic rhinitis the main indications for treatment are two: nasal stenosis and the excessive secretion and its retention.

The first indication can be met only by surgical measures, which consist in the removal of anterior and posterior turbinate hypertrophies, the correction of obstructive deflections of the septum, the evulsion of overgrowths of adenoid tissue in the pharyngeal vault, and the correction of local and general hypertrophies of the mucosa. The second main indication—namely, excess of secretion and its accumulation in the nasal cavities—is largely fulfilled when the foregoing procedures are successfully carried out, as upon the establishment of free nasal respiration and adequate nasal drainage, and the removal of the obstruction to the venous circulation constituted by the hypertrophies, the mucous membrane tends to, and frequently does with remarkable promptness, regain a more healthy condition. Systematic anterior and post-nasal douches (those with solutions of hydrogen dioxide being very efficient) and fine sprays of warmed vaselin are required to secure thorough cleanliness and restore the mucous membrane to its normal state.

In rhinitis atrophica the therapeutic management consists in, first, the removal of the incrustations and accumulated secretions; second, the maintenance of cleanliness of the nasal chambers; third, the control of the fœtor by means of deodorants and antiseptics; and, finally, the improvement of the general health and the energetic treatment of any specific dyscrasia that may exist.

It is needless to add that the successful treatment of cases of chronic rhinitis requires the technical knowledge and operative skill of the specialist.

AUTUMNAL CATARRH.

Definition.—An affection of the upper air-passages, characterized by irritability of the mucous membrane, with catarrhal and asthmatic manifestations, by the abruptness of the onset of the attack, which recurs annually at or near a fixed date in the spring, summer, or early autumn, and by its immediate cessation upon the patient's reaching certain localities or on the occurrence of frost.

SYNONYMS.—Hay or Rose cold; Hay-asthma; Hay-fever; Summer catarrh; Catarrhus æstivus; Periodic coryza.

Etiology.—The researches of J. N. Mackenzie have led to the discovery in early medical literature of evidences that this affection was occasionally, though imperfectly, recognized prior to the present century. To Bostock, however, is due the credit of having, first in 1819, and at greater length in 1828, accurately described it as a substantive disease. Within the past thirty years several treatises and innumerable journal articles have been written on the subject. The list of writers, whose contributions have especially attracted attention, includes the names of Murrill Wyman, Blackley, George M. Beard, Daly, Roe, Sajous, J. N. Mackenzie, Bosworth, and others. As the general outcome of the labors of these investigators it may now be affirmed that autumnal catarrh, popularly designated by the term "hay-fever," is provoked by certain stimuli, chief among which are mechanical irritants in the atmosphere acting upon a supersensitive nasal mucous membrane in individuals of neurotic temperament.

Most prominent among the exciting causes is unquestionably the pollen of various plants. This is, however, certainly not, as was for a long time thought, the only excitant of the attack. It is now known that inorganic dust of various kinds, the odors of certain flowers and other substances, the emanations from animals, as the horse, and from feathers, are capable of intensifying the symptoms during the attack and also of inducing similar symptoms at other seasons of the year. The intense glare of the summer sun, excessive heat, over-exertion, and indigestion commonly aggravate the attack. That these agencies are, as has been affirmed, in point of fact, exciting causes of the disease is questionable. Hay-fever and bronchial asthma are not only closely associated clinically, but they also resemble each other in respect of the causes by which the attack may be excited, and in the fact that there is a wide range of individual peculiarity in the reaction of different persons to the various stimuli.

The presence of local nasal trouble plays a most important part in the etiology of the disease. The resemblance to asthma in this respect is striking. Voltolini in 1871 reported the disappearance of asthma upon the removal of a nasal polyp, and the causal relation between nasal disease and bronchial asthma has come to be fully recognized. An analogous relationship between diseased conditions of the nasal mucous membrane and hay-fever has also been demonstrated. The lesions are those of hypertrophic rhinitis. There is not rarely deflection of the septum. Superadded to these is, however, the presence of an area of extreme hyperæsthesia in the nasal mucosa—hyperæsthesia often so exquisite that the touch of a probe will instantly excite the characteristic train of symptoms. This local sensitiveness is an almost constant factor in the etiology of the disease. Its frequent presence in those who do not suffer from autumnal catarrh proves, however, that something more is required. That "something" is the neurotic constitution. The peculiar nervous organization which constitutes this important predisposing factor frequently shows itself in several members of a family; it is transmissible by heredity. Its possession is a frequent misfortune of those living in affluence and refinement, but it is not restricted to any social class.

Hay-fever is more common in the United States and England than on the continent of Europe. In America the Anglo-Saxon population is more liable than others. Cases have been observed in the negro, though but rarely. Males suffer in greater proportion than females, and the disease may develop at any period in life, from childhood up. More than 33 per cent. of the cases declare themselves before the age of twenty years. Dwellers in cities are especially prone to the disease, but those who live in the suburbs and in villages, and even farmers in the open country, do not enjoy entire exemption. The earlier investigators of hay-fever in America were led to believe that its prevalence was chiefly restricted to the cities and to certain areas in the Eastern and Middle States. This opinion is now known to be without foundation. The affection may show itself in any part of the country when the peculiar irritants which excite it, and persons of neurotic constitution, with hyperæsthetic nasal mucous membranes, are found together. The atmosphere of agricultural regions aggravates the attack. There are certain localities in which the disease does not prevail. These localities are usually sharply defined, and possess in common the attribute of an uncultivated soil. They are mostly mountainous, as certain districts in the White Mountains, the Adirondacks, and the Catskills. But elevation is not the essential factor. Relief may be experienced in the Maine wilderness, at certain sea-shore places, at Beach Haven on the New Jersey coast, and on islands off the coast, as the Isles of Shoals and Nantucket. The advantages of any given locality are as uncertain and anomalous as the affection itself. The relief experienced by one sufferer is often wholly withheld from others; and a drive of a few miles in one case effects deliverance from suffering, while in another it brings back the symptoms in all their severity. If the patient has succeeded in finding the locality suited to his case, he may wholly escape the attack by repairing thither before its expected advent, and remaining until the term of its usual duration has expired. Should he during this period quit his refuge, the symptoms at once recur with full severity.

The discussion of the causation of hay-fever cannot be concluded without allusion to the prominence of the psychical element in many cases. In one case J. N. Mackenzie induced the attack by means of an artificial rose. In a large proportion of the cases recurrence of the attack takes place year after year, regardless of wind and weather, on a certain day of the month—a fact which has as yet found no rational explanation beyond the hypothesis of expectant attention. Certain it is that the high seas afford complete immunity, and a holiday trip to Europe constitutes a sovereign remedy.

Symptomatology.—The attack makes its annual return at or about the same date. There is in some of the cases a period of prodromes, which consist of lassitude and nervous irritability. The onset is abrupt. There is itching of the palate and throat—a most annoying symptom, and both common and characteristic. I have seen cases in which year after year this persistent itching constituted the only local symptom. Cases like these are, however, exceptional. Ordinarily the symptoms of an acute coryza develop rapidly, with great subjective disturbance. Frequent, uncontrollable sneezing; nasal obstruction; free rhinorrhœa, usually thin and watery, sometimes muco-purulent; great irritation of the eyes, with itching of the lids and lachrymation; loss of the sense of smell, impairment of that of taste, and not rarely disturbances of hearing, constitute the symptoms. These are intensified in paroxysms, and are likely to be aggravated by changes of temperature, by sunlight, and the open air. The accompanying constitutional disturbances consist of subjective sensations of heat and cold, great lassitude, complete loss of appetite, and sleeplessness. After a while the catarrhal derangement extends to the bronchi, and the patient is disturbed by cough; not rarely asthmatic symptoms are present, and add greatly to the distress of the patient. The symptoms vary in different cases, both in special localization and in intensity, and in the same persons in succeeding years. The whole duration of the attack, if not cut short by change of climate, is about six weeks. The autumnal cases usually continue until the appearance of decided frost, whereupon they at once cease.

The **prognosis** is favorable as regards recovery from any given attack and as regards length of life. Hay-fever does not end fatally, nor does it especially predispose to any form of chronic disease of the respiratory organs. The prognosis as regards the recurrence of the attack is much less hopeful. The recoveries reported have been ascribed, as a rule, both in children and in adults, to the successful local treatment of the nose. In many cases the recovery has not proved permanent.

Treatment.—The therapeutic indications are the neurotic constitution of the sufferer, the hyperæsthetic nasal mucosa, and the exposure to the atmospheric irritant which acts as the exciting cause of the attack.

The first of these indications is to be met by favorable hygienic conditions, diminished physical and mental strain, orderly living, and the use of those drugs which experience has shown to exert a favorable influence upon the nervous system. Among these are arsenic, strychnine, phosphorus, and quinine. The second indication is fulfilled by the efficient local treatment of any nasal trouble that may exist, and especially by the destruction, by means of chemical agents or by the galvano-cautery, of the mucous membrane, with its terminal nerve-filaments, in the areas found to be hyperæsthetic, and to develop catarrhal symptoms in response to the touch of a probe. The procedures by which this is accomplished should be entrusted to the specialist. Third, patients should spend the period of the autumnal attack in a favorable locality, either in the dry air of the mountains or by the sea. It is a fortunate thing that by far the greater number of hay-fever patients belong to the favored class, with whom a six-weeks' summer vacation is neither unusual nor impracticable.

EPISTAXIS.

Definition.—Bleeding from the nasal passages.

SYNONYM.—Nose-bleed.

Etiology.—Bleeding from the nose may be due to local or constitutional causes. It is sometimes due to a combination of both, as in children, in whom it is favored by the great vascularity of the nasal mucous membrane, by the frequent presence of "hæmorrhagic spots," and the erosions of the septum produced by picking the nose, but in whom some active congestion or general disturbance of the circulation is required to bring it about. Nose-bleed in children is a common and unusually insignificant phenomenon. Local causes of nose-bleed are, in addition to the hæmorrhagic spots and erosions mentioned, chronic rhinitis, intranasal ulceration, new growths, the presence of foreign bodies, and various kinds of traumatism, especially contusions of the face.

In fractures involving the bones of the face and cranium blood may escape from the accessory sinuses or from the middle ear by way of the nose, or in hæmorrhage from the lungs or œsophagus or stomach some part of the blood may be discharged from the nose. In the case of fracture, hæmorrhage through the nose may be profuse, and even fatal. These blood-losses not from, but merely by way of, the nose do not in the strict sense constitute epistaxis—a term restricted by systematic writers to hæmorrhage having its origin within the nasal passages.

Among the constitutional causes are exposure to extreme cold or undue heat, or to a rarefied atmosphere as in the ascent of high mountains and in balloon ascensions. It occurs with frequency both in boys and girls at the age of puberty. It may result from the suppression of the menstrual flow or follow the sudden arrest of a customary hæmorrhoidal discharge. It is of frequent occurrence in those suffering from anæmia in its various forms, and is common in persons of plethoric habit. The tendency to nose-bleed is in certain families hereditary. In hæmophilia nose-bleed constitutes a very frequent manifestation of the hæmorrhagic diathesis. It is a common symptom also in scurvy and purpura, and occurs in erysipelas, the malarial and the malignant fevers, and in nasal diphtheria. In the last it may take the form of a continuous oozing or a copious discharge, repeated at intervals during the day. Nose-bleed in diphtheria is a symptom of grave import. Slight nose-bleed occurs in the first week of typhoid fever with such frequency as to acquire diagnostic importance. It often amounts to a few drops merely, and may be overlooked by the patient as well as by his attendants. Exceptionally it is of moderate amount. Nose-bleed not infrequently results from the congestion and shock of the violent convulsive cough of pertussis. It is by no means a rare symptom in advanced disease of the kidneys and in various affections of the liver. It is worthy of notice that in the venous engorgement of cardiac and pulmonary diseases, even with marked cyanosis, nose-bleed is infrequent. Finally, it may result from violent mental emotions.

When epistaxis is due to general causes the blood escapes by capillary oozing from one, rarely two or three, limited areas of the respiratory portion of the cartilaginous septum, and in most instances it proceeds from one side only. In a very small proportion of the cases it comes from the turbinated bodies or from the floor of the nostril. The mucous membrane is deeply congested, of a violaceous-red color, and shows minute spots of ecchymosis.

Symptomatology.—Epistaxis is sometimes heralded by prodromes, consisting of giddiness, pressure in the head, and a sensation of dryness, tickling, or obstruction in the nostrils, which impels the patient to more or less forcibly blow the nose. More frequently these symptoms are wanting, the bleeding occurring suddenly and without warning. The blood may flow in drops or for a time in a continuous stream. Ordinary, slight nose-bleed generally ceases in from fifteen to twenty minutes, and is without immediate clinical importance, whatever may be found to be its remote significance. The graver bleedings may be protracted for hours or days, and, while a fatal case is of rare occurrence, serious consequences are likely to follow profuse hæmorrhage. The arrest takes place by clotting at the point of oozing. It is important to examine the pharynx, as the clot in the nostril may lead to the escape of blood by way of the posterior nares, and its being swallowed. The vomiting of blood thus swallowed may be mistaken for hæmatemesis; its expulsion by cough, for hæmoptysis; but not if due care be observed in the investigation of the case.

Treatment.—The cessation of the bleeding results from the formation of a clot, and the tendency to clotting increases with the blood-loss. In the majority of the cases the hæmorrhage ceases spontaneously in the course of a little time. Rest is important. Fear and excitement should be allayed. The patient should breathe through the mouth and refrain from attempts to expel clots. Ice may be applied to the nose or to the nape of the neck, and the hands held over the head. If the bleeding do not cease, the alæ nasi may be compressed and clotting favored by the gentle injection of water, either cold or as hot as can be comfortably borne. Tannic acid, in powder or solution, usually acts with promptness, and a solution of cocaine, of from 2 to 5 grains to the ounce, either as a spray or applied by means of dossils of cotton, is frequently efficient. If the foregoing measures are unsuccessful, the nasal cavity should be treated with iodoform and packed with strips of antiseptic gauze. The perchloride of iron in solution and the solution of the subsulphate of iron are styptics of great value. Bleeding from an ulcerated surface may be arrested by chemical cauterants, as chromic acid, or by the galvano-cautery. Plugging of the posterior nares should only be resorted to as an extreme measure. It can be rapidly done by means of a soft catheter, a far safer, cheaper, and more convenient instrument than that of Bellocq. The ready method of Levis consists in pushing, by means of a probe or grooved director, a small piece of fine sponge, to which is tied a stout string, along the floor of the nostril to the naso-pharynx, threading a series of small sponge disks upon the string, pushing them gently in succession into the nose, and tying on a larger piece to occlude the anterior nasal meatus. Before plugging the nares the nasal cavity should be cleansed with an antiseptic solution and freely treated with iodoform. In stubborn nose-bleed ergot or ergotine and opium or morphine may be required, and should preferably be administered by the hypodermic method.

DISEASES OF THE LARYNX.

BY JAMES C. WILSON.

CATARRHAL LARYNGITIS.

Definition.—Catarrhal inflammation of the mucous membrane of the larynx.

SYNONYM.—Laryngeal catarrh.

Catarrhal laryngitis may be—(1) acute; (2) subacute; or (3) chronic.

1. Acute Laryngitis.

Etiology.—"Taking cold," prolonged exposure to a cold, damp atmosphere, over-use of the voice in speaking, shouting, or singing, especially under unfavorable atmospheric conditions, as in crowded and badly-ventilated halls, are common causes of acute laryngitis. It may be produced by the inhalation of air charged with smoke or irritating gases or vapors. Less frequently it is due to traumatism caused by the lodgement of foreign bodies, the action of very hot liquids or corrosive poisons, or external violence. It also occurs as a local manifestation of the general catarrhal process in measles, influenza, and variola, and as a complication in other acute infectious diseases, as scarlet fever, enteric fever, and erysipelas.

Catarrh of the larynx is very often associated with catarrh of the naso-pharynx and bronchi.

The predisposition to laryngitis varies greatly in different families and individuals.

Symptomatology.—There is a disagreeable sensation of dryness and tickling in the throat; the inspiration of cold air and efforts to converse cause pain. Laryngeal tenderness is experienced upon external pressure. Spontaneous pain is, however, moderate or wholly absent.

Cough is present. It is tickling and hoarse, or "laryngeal," in character; at first dry, later attended with scanty muco-purulent expectoration, which in severe cases may be slightly streaked with blood.

The voice is changed. At first husky, it grows rapidly hoarse, and at length may be completely lost. The degree of hoarseness varies in different cases, according as it is due to changes in the secretions of the larynx, to swelling of the mucous membrane, or to paresis of the vocal cords from inflammatory infiltration of the muscles. Dyspnœa is not common in adults;

it is, however, a very frequent symptom in early life, when it is likely to occur in paroxysms and at night.

In severe cases of acute laryngitis cough is very harassing, deglutition is painful, and there may be urgent dyspnœa.

Inspection by means of the laryngoscope reveals those changes which are seen in catarrhal inflammation elsewhere. The mucous membrane is reddened and swollen, especially between the arytenoid cartilages and in the ary-epiglottic folds. The vocal cords are no longer smooth and glistening, but appear swollen and more or less reddened. When the inflammation is intense they present superficial erosions, and minute hæmorrhages are seen at various points of the laryngeal mucous membrane. A scanty exudation of altered mucus adheres irregularly to the surface. These changes vary in degree in different cases, and in various parts of the larynx in the same case. In phonation there may often be observed imperfect approximation of the vocal cords, due to implication of the intrinsic muscles of the larynx in the inflammatory process.

The constitutional symptoms vary. As a rule, they are not severe; moderate fever, with headache, loss of appetite, and debility, may, however, occur.

Acute laryngitis lasts from a few days to a week or more, and terminates in recovery. Neglected cases are liable to run into the chronic form.

Acute Laryngitis of Children.

SYNONYMS.—Spasmodic croup; False croup.

As it occurs in young children this affection deserves separate consideration. The special feature consists in paroxysmal exacerbations, suffocative in character and occurring at night. These are to be ascribed to the relative smallness of the larynx in infancy, the narrowness of the rima, the looseness and vascularity of the mucous membrane, and the greater reflex excitability of the nervous system. The disease is a familiar one, occurring with greatest frequency during the first dentition, and particularly during the second and third years; after the fifth year it is less common, though cases occur up to the age of puberty.

Etiology.—Exposure to cold and damp, sudden chilling of the surface, prolonged and violent screaming, the inhalation of steam, smoke, and dust, the gastric catarrh resulting from an attack of indigestion, are determining causes of acute laryngitis in infants. It is much more frequently seen in the cold, damp months of winter and spring than in the summer and autumn. It occurs somewhat more commonly in male than in female children, and certain families and individuals show a marked liability to recurrence—a liability that is in some instances hereditary.

Symptomatology.—During the day the child has been in usual health, or may have coughed a little and have been somewhat feverish and out of sorts, without being positively ill. At bed-time the cough has the ringing, metallic character known as croupy, but the patient goes to sleep as usual. About midnight or shortly thereafter he awakes suddenly with oppressed breathing, huskiness of the voice or complete aphonia, and a harsh, croupy cough. Inspiration is prolonged and stridulous; there is recession of the suprasternal and supraclavicular spaces; the pulse is frequent and small; and the lips and finger-tips are cyanotic. There is great restlessness, and the expression of the little patient's face indicates anxiety and distress. The attack presently passes off, either spontaneously or as a result of the administration of simple remedies. The child presently falls asleep again, and, with the exception of some uneasiness and occasional cough, rests until morning; or the attack may be repeated once or several times in the course of the night. On the following day he scarcely seems ill, and plays about as usual; but toward evening the croupy cough reappears, and the next night the attacks of croup occur as before, to be again repeated, as a rule, upon the third, and rarely the fourth, night, but with diminishing severity. After that there remains simply a trifling bronchial catarrh, which in the course of a few days disappears.

The sudden onset and rapid subsidence of these severe suffocative attacks suggest their origin in the nervous system. But that they are wholly due to reflex laryngeal spasm is very doubtful. Sudden swelling of the mucous membrane and the accumulation of secretion during sleep also play an important part in their etiology.

Diagnosis.—Acute laryngitis of the adult rarely presents difficulty in diagnosis. The severer cases suggest œdema of the larynx (acute laryngeal œdema), while those that are attended by complete loss of voice may be mistaken for hysterical aphonia or paralysis of the vocal cords due to other causes. These questions are at once settled by the laryngoscope.

In children the diagnosis of acute laryngitis is in certain cases attended with serious difficulty. The condition is to be distinguished from laryngismus stridulus by the presence of fever, the catarrhal symptoms, the mode of onset, the character of the paroxysms, their nocturnal occurrence, the preceding hoarseness and loss of voice, the absence of the prolonged crowing inspiration which terminates the attack of laryngismus, and the course and duration of the disease.

The differential diagnosis between spasmodic croup and laryngeal diphtheria (membranous croup) may for a time be impossible. The principal points in favor of spasmodic croup are the milder character of the constitutional symptoms which precede the signs of laryngeal obstruction, the paroxysmal nature of the obstruction, and the complete relief between the attacks; the progressive amelioration of the symptoms after the second night; the absence of exudation upon the tonsils and adjacent parts, and the absence of enlargement of the cervical lymphatic glands.

Treatment.—In severe cases the patient should remain in his room or bed. The use of the voice should be proscribed. The room should be kept at an equable temperature of about 70° F., and cautiously ventilated. The inhalation of steam from any simple apparatus gives relief and has a favorable influence upon the course of the attack. Bits of ice allowed to melt in the throat are useful; so also is the constant application of an ice-bag externally. When the process is active and the symptoms urgent, the application of leeches over the larynx may become necessary. The treatment by drugs is symptomatic: for the relief of the distressing cough, morphine, gr. $\frac{1}{12}$–$\frac{1}{6}$, *p. r. n.*, or Dover's powder, gr. iij–v, every three or four hours, may be administered. The diet should be of the most simple kind, consisting chiefly of gruels, arrowroot, eggs, bland broths, and milk. Warm milk or equal parts of milk and Vichy, warmed, are excellent. For milder cases a less vigorous course suffices.

The treatment of spasmodic croup consists in the employment of measures to arrest the paroxysm and to prevent its recurrence. Among the former are a prompt emetic of ipecac in the form of the wine or syrup; a hot bath; the application over the larynx and upper part of the chest of a sponge dipped in hot water; or, if the dyspnœa be urgent and not immediately relieved by these means, the cautious administration of a whiff or two of chloroform. The following prove useful in mitigating or preventing the recurrence of the paroxysm: Small doses of ipecac at intervals during the day; gentle purging by means of calomel or castor oil; a dose of Dover's powder proportionate to the age of the child, either alone or in combination with a minute quantity of tartar emetic at bed-time and repeated in the course of two or three hours. It is well to keep the child in bed during the day following the attack, and to have the air of the apartment moistened by steam. Children who have shown a predisposition to acute laryngitis should have the neck and throat bathed daily with cold water, followed by brisk friction, and should be well clothed in flannels to the ankles.

2. Subacute Laryngitis.

By far the larger number of cases of catarrhal laryngitis are of the mildest type. The patients are not ill at all. The only symptoms are a slight tickling cough, with hoarseness, passing to loss of voice, and accompanied by pain or prolonged tickling.

The **etiology** comprises the same causal factors as that of the acute form, acting with less intensity; and the morbid appearances as revealed by the laryngoscope are those of a catarrhal inflammation of mild form. The condition acquires importance from its great relative frequency; from the fact that, being accompanied by trifling subjective symptoms, it is likely to be neglected; and, finally, because in many cases prolonged, habitual exposure to the original cause, or use of the voice when the larynx is slightly congested or inflamed, converts a passing local indisposition into a serious disease. In point of fact, the larger proportion of cases of chronic laryngitis arise in this way.

The **treatment** of subacute laryngitis consists in the avoidance of all conditions which are liable to excite laryngeal inflammation, in rest of the voice as nearly complete as possible, and the occasional inhalation of steam. Tablet-triturates of morphine, gr. $\frac{1}{24}$–$\frac{1}{16}$, or of Tully's powder (pulvis morphinæ compositus, U. S. P.), gr. ij–iij, allowed to break up slowly at the back of the mouth, at intervals of an hour or more, exert a favorable influence upon the course of the attack. The value of gargles and washes and potassium chlorate in the treatment of this condition has been over-estimated. Local applications to the larynx by means of applicators and the like are wholly unnecessary.

3. *Chronic Laryngitis.*

Etiology.—This form of catarrhal laryngitis may be the sequel of an acute attack; more commonly it is the result of the persistent action of causes which give rise to subacute catarrh. Improper use of the voice and its habitual over-use in singing, public speaking, or shouting in the open air are very common causes of chronic laryngitis. It occurs also in connection with chronic pharyngitis, and especially with that form which is produced by habitual over-indulgence in alcohol and tobacco; in certain cases of marked obstruction to nasal respiration, and in cases of elongation of the uvula.

Chronic laryngitis is more common in males than in females, and is especially a disease of middle life.

Symptomatology.—There is a more or less constant tickling sensation in the throat, accompanied by a desire to obtain relief by coughing; as a rule, pain is not present, except after prolonged coughing or efforts to talk. Many patients complain of a disagreeable feeling of dryness. The voice is rough and hoarse, and at times almost lost. The cough is ringing, loud, deep; expectoration is, as a rule, scanty and tenacious, but occasionally abundant, and sometimes fœtid. Upon laryngoscopic examination the mucous membrane is seen to be irregularly thickened and discolored, but the redness is less intense than in the acute form. The vocal cords are of a grayish-red color, and in debilitated and cachectic persons there may be discovered minute superficial erosions. The epiglottis is in many cases irregularly thickened.

The general health is often impaired, although there are no constitutional symptoms peculiar to this affection.

Diagnosis.—The local morbid sensations, chronic alteration of the voice, and the peculiar cough will lead us to suspect the true nature of the affection, but a positive diagnosis can only be made after careful laryngoscopic examination. In every case of chronic laryngitis the history of the patient in all particulars must be carefully investigated, and the condition of the lungs and other organs carefully examined, in order to determine whether or not the local affection be primary, or secondary to some other disease, as alcoholism, tuberculosis, or syphilis.

Treatment.—An effort should be made to remove the patient from those injurious influences which have brought about the catarrh. To this end temporary, or even permanent, change of climate and occupation may be necessary. Attention must also be given to his personal habits, especially as regards the use of tobacco and alcohol, and to general hygiene and dietetics. Ill-ventilated and overheated rooms are to be avoided; also undue use of the voice. The throat and chest should be daily sponged with cold water. Chest-protectors and thick scarfs are injurious. Constitutional treatment must be instituted when the general health is at fault; nor is judicious internal medication in all cases without favorable effect upon the local malady. The prolonged administration of cubebs, tar, benzoin, ammonium chloride, arsenic, and the preparations of iodine has been followed by good results. The patient must be instructed in the use of sprays, which he should regularly employ at intervals during the day. To this end proper solutions of ammonium chloride, sodium chloride, sodium biborate, boric acid, sodium bicarbonate, and sodium or potassium iodide may be advantageously employed. Should astringents be indicated, weak solutions of alum, tannic acid, zinc sulphate, or silver nitrate may be used. The physician should make intralaryngeal applications at proper intervals. Among those agents which exert a favorable influence are silver nitrate, zinc sulphate, and tannic acid in solutions of varying strength; bismuth subnitrate, iodoform, aristol, and powdered catechu by insufflation, and a 20-grain solution of menthol in olive oil.

Œdematous Laryngitis.

Synonyms.—Acute laryngeal œdema; Œdema of the glottis.

Etiology.—Œdema of the mucous and submucous tissues of the larynx occasionally occurs as a serious and frequently fatal complication in the course of acute catarrhal laryngitis, whether due to cold or to internal or external traumatism; in the course of chronic diseases of the larynx, as tuberculosis and syphilis; in connection with perichondritis of the larynx; in connection with severe inflammatory affections of neighboring structures, as the tonsils, parotid glands, and the cellular tissue of the neck; in the course of acute infectious diseases, as scarlatina, typhoid fever, variola, and erysipelas; and, finally, as an extension of the general œdema of acute or chronic nephritis.

Symptoms.—Rapidly progressive dyspnœa is the chief symptom. It is at first inspiratory; later, also expiratory. Respiration is often accompanied by loud stridor. The voice becomes husky, and soon fails. Signs of impending suffocation supervene, and unless relief is afforded death takes place in the course of a few hours. If a laryngoscopic examination prove successful, the epiglottic and ary-epiglottic folds are seen to be greatly swollen, the latter almost meeting laterally; the false cords are also œdematous. These changes can be felt with the finger, and upon depressing the tongue the swollen rim of the epiglottis may sometimes be brought into view.

The **diagnosis** is unattended with difficulty, and depends upon physical exploration.

Treatment.—An ice-bag externally and ice internally may arrest the œdema. The free administration of the bromides has been advised. In suitable cases leeches may be applied. If these measures fail to give prompt relief, the throat may be sprayed with a solution of cocaine and the œdematous parts scarified. If the evidences of obstruction persist, immediate recourse to tracheotomy is necessary to save life.

Pseudo-membranous Laryngitis.

Definition.—Inflammation of the mucous membrane of the larynx, resulting in the formation of a pseudo-membrane or pellicle composed of a network of fibrin, embracing in its meshes leucocytes and necrotic epithelium.

SYNONYMS.—True croup; Membranous croup; Fibrinous laryngitis; Laryngeal diphtheria.

Etiology.—Any agent capable of destroying the protecting epithelium of the laryngeal mucous membrane, and thus permitting the escape of serum and white blood-corpuscles, may give rise to the formation of a pseudo-membrane. Hence this form of laryngitis may result (*a*) from traumatism, as the inhalation of steam, hot smoke, or irritating and corrosive chemicals in the form of vapor or solution; and (*b*) from the action of certain pathogenic micro-organisms. Among the latter are a streptococcus demonstrated to be biologically identical with the streptococcus pyogenes and the Klebs-Loeffler bacillus of diphtheria. Traumatic membranous laryngitis is not included in the following account.

Pseudo-membranous laryngitis occurs at all seasons of the year. It especially affects young children between the ages of two and six. Cases in children under two and over seven years of age are much less common. Exceptionally, however, the disease occurs at a later period of life. Boys are somewhat more liable than girls. This affection frequently occurs as a complication in scarlet fever and measles, in which case it is, as a rule, secondary to pseudo-membranous inflammation of the tonsils or adjacent structures. It occurs, however, in by far the greater number of cases as a manifestation of diphtheria, secondarily by extension, and much less frequently as a primary manifestation.

So overwhelming is the preponderance of the unquestionably diphtheritic cases that many competent observers regard all the cases as of that nature—an extreme view that finds ample justification in the fact that no criteria have yet been established by which the differental diagnosis between diphtheritic pseudo-membranous laryngitis and the so-called non-contagious membranous croup of the dualists can be made by the clinician during life or by the pathologist after death, save only the presence or absence in the exudate of the Klebs-Loeffler bacillus. In both the symptoms are the same; the course and termination of the attack are not different. If it be urged that the cases to which the name "croup" is applicable are of milder intensity, that they occur sporadically, and that they do not appear to be contagious, the reply may be made that cases of undoubted tonsillar diphtheria are also occasionally met with that develop in localities previously free from the disease and in the absence of traceable contagion, which run a favorable course, and which do not transmit the disease to exposed and apparently susceptible individuals. But no one, therefore, denies the specific nature or the contagiousness of diphtheria. Physicians who look upon every case of pseudo-membranous laryngitis as a probable case of diphtheria enjoy the practical advantage of erring, if err they do, upon the safe side. Such a working hypothesis impels to promptness and energy in treatment, and especially in prophylaxis, which often results directly and indirectly in the saving of life. Those, on the other hand, who, governed by tradition and authority, regard these cases as instances of membranous croup, to their minds neither specific nor contagious, too often expiate in the toils and sorrows of a virulent local epidemic their devotion to a dogma unsupported by facts.

For the full consideration of Diphtheria as an acute infectious disease the reader is referred to the subject under its appropriate heading.

Morbid Anatomy.—The pseudo-membrane in the larynx constitutes the essential lesion. It varies greatly in amount and distribution. It may involve the mucous membrane uniformly or only here and there in patches. It often covers the concavity of the epiglottis, the ary-epiglottic folds, the false cords, extending deeply into the ventricles, the true cords, and it may extend into the trachea and remotely into the finest bronchi. It is usually associated with a similar exudate in the pharynx or on the tonsils. In cases in which no visible membrane has been detected on these parts during life it has frequently been demonstrated post-mortem upon the posterior pillars of the fauces and the posterior surfaces of the tonsils. The consistency of the pseudo-membrane is variable. It may be thin and friable, or thick, tough, and tenacious. It sometimes appears as a thick, membranous mass of superimposed strata. In color it is usually of a dirty yellowish-gray. The surrounding and underlying tissues are more or less swollen, deeply congested, and infiltrated with leucocytes. Upon membrane covered with pavement epithelium the exudate takes a deep and firm hold, implicating not only the subepithelial, but also, in many instances, the submucous tissues, which are involved in the necrotic process; but on mucous membranes covered with columnar ciliated epithelium the fibrinous material is commonly found to be rather superimposed, and to have involved principally the epithelium, without destroying the underlying tissues. For this reason the pseudo-membrane in faucial diphtheria is removed with difficulty, leaving during life a bleeding and ulcerated surface, and after death a deep solution of continuity; whereas in laryngeal and tracheo-bronchial diphtheria it may be readily stripped from the surface, leaving exposed a swollen and deeply injected, but unbroken, mucous membrane.

Like the mucous membrane of the rest of the air-passages, that of the larynx is covered in the greater part of its extent with a columnar ciliated epithelium. . . . . The cilia are found higher up in front than on each side and behind, reaching in the former direction as high as the highest portion of the epiglottis, and in the other directions to a line or two above the superior vocal cords; above these points the epithelium loses its cilia and gradually assumes a squamous form, like that of the pharynx and mouth. Upon the vocal cords the epithelium is squamous, although both above and below these it is ciliated (Quain).

Whether the pseudo-membrane be *croupous*—that is, formed upon the surface and detachable—or *diphtheritic*—that is, implicating the mucous and even the submucous tissues, and structurally adherent—is not so much determined, as is frequently thought, by the intensity of the inflammatory process as by the character of the tissues involved. Hence there is no necessary etiological difference between a croupous and a diphtheritic inflammation; on the contrary, the action of the same agent may produce both forms in different but adjacent mucous membranes. The German pathologists are therefore correct in speaking of the process as a *croupous-diphtheritic* inflammation.

Symptomatology.—The symptoms usually develop in the course of an attack of faucial diphtheria or of one of the exanthemata. Less frequently they arise as the manifestations of a primary laryngeal diphtheria. They point to progressive impairment of the functions of the larynx, with increasing obstruction to respiration and its consequences, and consist of hoarseness, explosive and croupy cough, stridulous respiration, dyspnœa, recessions, restlessness, cyanosis, and stupor.

Hoarseness is, as in acute catarrhal laryngitis, the earliest symptom. It is attended by cough, and the voice is generally reduced to a whisper, or is even completely lost.

The cough is paroxysmal—at first explosive, like that caused by the accidental lodgment of a foreign body in the larynx. Without wholly losing this explosive character it becomes hoarse, then metallic, and finally, as the dyspnœa grows more urgent, dry and whistling.

In the course of some hours, sometimes not for a day or two, the signs of decided obstruction to the respiration manifest themselves. Difficulty in breathing is at first paroxysmal, and followed by intervals of partial relief; it soon, however, becomes continuous. The respiration is now stridulous, inspiration first, and after a time expiration, assuming this character. The stridor, in the beginning low-pitched and hoarse, gradually and concurrently with similar changes in the cough becomes high-pitched and metallic. Respiration is now hurried and labored, and accompanied by loud, dry, and whistling sounds, which are interrupted from time to time by harassing and for the most part unproductive cough. Occasionally, however, in the more severe paroxysms, fragments of membrane become detached and are coughed out.

At this stage of laryngeal obstruction the inspiratory recessions of the more yielding portions of the thorax constitute an important and suggestive sign. They are marked in the suprasternal and supraclavicular regions, at the lower portion of the thorax, and finally, when the obstruction has attained a high grade, the cartilages yield and inspiratory recession of the sternum takes place.

Meanwhile, the progressively deficient oxygenation of the blood gives rise to characteristic effects upon the nervous system. There is extreme and distressing restlessness; the child tosses from side to side in his struggle for breath; his expression is anxious and frightened. His color, which has for some time been pale and leaden, with slight lividity of the lips and finger-nails, now becomes continuously cyanotic, the face dusky, the lips blue, the hands mottled, the nails dark. The restlessness presently gives place to stupor, which passes into a gradually deepening coma and ends in death.

Complications.—Diphtheria of the larynx shows a marked tendency to spread to neighboring parts. Cases in which upon post-mortem examination the pseudo-membrane has not extended beyond the larynx are very rare. On the contrary, it is often seen to invade rapidly and successively the adjacent surfaces in an upward direction, sometimes extending even to the nostrils —nasal diphtheria. Much more commonly, however, its extension is downward. The mucous membrane of the trachea, and of the bronchi in some cases even to their most remote subdivisions, is in turn involved; or, owing to the prolonged inspiratory efforts, the obstruction to expiration, and the general enfeeblement of the respiratory powers, shreds of membrane or masses of secretion are drawn down deeply into the lungs, and there set up inflammation of the finest bronchial twigs and the associated vesicular structure.

Pseudo-membranous tracheo-bronchitis and more or less extensive bronchopneumonia constitute, therefore, the two more important complications of this form of laryngitis. Their development is gradual, and marked not only by an aggravation of the general symptoms, but also, usually though not invariably, by the development of the characteristic signs of their respective lesions. It is of great practical importance to recognize when possible the presence of these complications, seeing that they gravely affect the prognosis and constitute the cause of death in a majority of the cases which terminate fatally after the performance of intubation or tracheotomy.

Râles are heard upon auscultation, and by their character and distribution indicate the nature and extent of the pulmonary lesions. They are much modified by the degree of laryngeal stenosis, the feebleness of respiration, and the loud and prolonged stridor. In estimating their significance it is therefore necessary to make due allowance for these disturbing factors.

Diagnosis.—Acute progressive laryngeal stenosis in a young child is nearly always due to pseudo-membranous laryngitis. If traces of the exudate can be discovered upon inspection of the throat, or if upon physical examination there can be detected coarse or whistling tracheal râles, or, finally, if shreds of membrane are expectorated after paroxysms of explosive cough, the diagnosis becomes sure. It is equally so, in the absence of such confirmatory evidence, if the case occur in a locality already the scene of an epidemic of diphtheria. The fact that even in pseudo-membranous laryngitis the signs of obstruction are at first paroxysmal, and followed by intervals of partial relief, must always be borne in mind. For this reason the early differential diagnosis between this disease and spasmodic laryngitis is not in all instances possible. In the latter, however, the intervals of relief are more complete and prolonged, the paroxysm not usually recurring until the succeeding night; the tendency is to progressive amelioration of the symptoms rather than progressive aggravation, and the signs of grave constitutional disturbance do not show themselves.

Prognosis.—Pseudo-membranous laryngitis is an extremely fatal disease. According to O'Dwyer, one child in ten with well-marked symptoms of laryngeal diphtheria recovers under medical treatment. Under all forms of treatment the mortality ranges from 60 to 80 per cent. The statistics of Stern show 26.4 per cent. of recoveries after intubation, and 26.5 per cent. of recoveries after tracheotomy. The fatal issue may be due to a very limited exudation. The extension of the process downward into the bronchi or the development of extensive broncho-pneumonia renders the prognosis in the highest degree unfavorable. It is especially gloomy in children under two years of age.

Treatment.—Alike in primary and secondary cases the treatment should be that of diphtheria. Even in cases in which the early differential diagnosis between spasmodic and pseudo-membranous laryngitis is not yet possible, the physician will act prudently and may save time by adopting this course. Two drugs are at present in favor—mercury and iron. Mercury may be given as calomel or as the bichloride. Children manifest a remarkable tolerance for mercury in diphtheria. Calomel may be administered to a child three years old in doses of from one-fourth to one grain every hour or every two hours, or the bichloride in doses of one-fortieth of a grain at the same intervals, and these doses continued for two or even three days, the medicament being guarded, if the bowels move too freely or mucus appears in the stools, by minute doses of opium in the form of paregoric or Dover's powder. The bichloride must be well diluted, and it is advisable to precede each dose by a draught of water. Upon the appearance of symptoms indicating relief of the laryngeal stenosis the dose is to be gradually decreased and the intervals prolonged.

Iron is best given in the form of the tincture of the chloride, the dose for a child of three years being from 3 to 5 drops in a teaspoonful of sweetened water every hour. Larger doses are liable to occasion disturbance of the stomach.

Alcohol in the form of whiskey or brandy, well diluted, is to be given in all cases from the outset, its effect upon the pulse, the first sound of the heart, and the nervous system constituting the guide for the regulation of the amount.

The diet should consist chiefly of milk, which should be given freely, either alone or diluted with Vichy water, and preferably warm. To this may be added at intervals such concentrated and easily assimilated articles of food as meat-juice, chicken-jelly, or the various commercial peptones and peptonoids. Water must be freely given. From the beginning of the attack the nasal passages and the pharynx must be systematically sprayed with a 10-volume solution of hydrogen dioxide diluted with an equal quantity of water, or with a solution of bichloride of mercury, 1 : 5000, with the object of retarding the upward extension of the inflammatory process. Efforts to act upon the intralaryngeal exudate by means of sprays are not often successful, and direct intralaryngeal applications by other means are impracticable. Furthermore, the attempt to make such applications is, in a struggling child, liable to damage and abrade mucous surfaces not yet affected, and thus favor the extension of the pseudo-membrane. A fine continuous spray of lime-water directed toward the child's mouth is often useful. From the beginning the patient should continuously inhale an atmosphere charged with steam. To this end a tent or canopy of blankets or other suitable material is arranged over the crib or cot, a large opening being left at the side near the head for ventilation, and steam being introduced at the middle by means of a tin pipe or "leader" from a croup-kettle. A tablespoonful of turpentine and a few drops of oil of eucalyptus may be added to the water in the kettle every three or four hours. I have seen the use of alcohol in the water, in the proportion of one part to seven or eight, followed by mitigation of the dyspnœa and relief of restlessness.

Along with the steam, oxygen may from time to time be introduced into the tent near the patient's face by means of a rubber tube.

Ice-bags and hot poultices are frequently applied to the throat. The latter are often agreeable to the patient. Both of these applications constitute an additional impediment, slight though it be, to the respiration, and neither is attended by demonstrable benefit sufficient to justify its employment.

An emetic is very often followed by temporary relief, and occasionally results in the expulsion of large masses of false membrane. It sometimes happens that the relief thus afforded is permanent, and marks the onset of convalescence. For this purpose the yellow subsulphate of mercury, in doses of from 2 to 5 grains, is most used. It is, as a rule, prompt and efficient, but it is open to the objection that if it fail to cause vomiting it is liable to set up a serious gastro-enteritis. For this reason in case of failure the dose should not be repeated, but an attempt to induce vomiting by ipecac should be made instead. In the course of a few hours, if indicated by the symptoms and warranted by the strength of the child, the emetic should be repeated. Too much time must not, however, be consumed in futile attempts to overcome a mechanical obstruction to breathing that does not yield to the foregoing measures. The question of surgical interference by intubation or tracheotomy must be laid before the parents of the patient early in the case, in order that it may receive in advance full consideration. Their consent to intubation will be obtained much more readily than to tracheotomy. The prospect of success is greater in proportion as the operation is performed early. The stereotyped objection, that cases in which operation has been refused have occasionally recovered without it, is more than offset by the fact that many cases that might have been saved by an early operation have been lost by delay. When the symptoms and physical signs indicate an extension of the false membrane into the bronchi, an operation should not be advised, except with the full understanding that it is undertaken as a forlorn hope.

The general management of the case is of the utmost importance. Much depends upon the skill and judgment of the attendants. Moments occur, especially after operation, when coolness and promptness of action avert impending suffocation. The patient must be carefully isolated, and those measures of personal and local disinfection strictly carried out which are employed in the management of the other contagious diseases.

TUBERCULOUS LARYNGITIS.

Definition.—Inflammation of the tissues of the larynx caused by local tuberculosis.

SYNONYMS.—Laryngeal phthisis; Throat consumption.

Etiology.—After prolonged discussion it is now generally conceded that tuberculosis of the larynx may occur as a primary disease. Much more frequently, however, it is secondary to pulmonary tuberculosis. When the earliest symptoms are laryngeal, the disease remains for a time localized, but eventually the lungs become involved. Secondary tuberculous laryngitis, if all cases are included, occurs in upward of 25 per cent. of the pulmonary cases. The laryngeal symptoms are, however, pronounced and the lesions extensive and advanced in a much smaller percentage. A majority of the cases occur in males—a fact attributed to their greater liability to chronic catarrhal laryngitis, which acts as a predisposing cause. Whether the affection occurs as a primary or secondary lesion, it is in either case due to local infection—in the first by means of tubercle-bacilli conveyed by means of the inspired air; in the latter, by the sputum.

Morbid Anatomy.—Not every case of chronic laryngitis associated with pulmonary tuberculosis is necessarily tuberculous. The mechanical irritation of frequent and severe cough and the contact of the sputum may cause chronic catarrhal laryngitis, which is aggravated by the condition of the patient, and which undoubtedly, after a time, predisposes to infection. In the tuberculous cases the mucous membrane is of a grayish, pale color, irregularly mottled and congested; it is at first swollen and studded with miliary tubercles, which by their coalescence form scattered tuberculous nodules. These nodules undergo caseation, as a result of which there form more or less extensive superficial ulcers, which show a tendency to spread. The floor of these ulcers is covered by a grayish exudation, and they are surrounded by a border of infiltrated and swollen tissue. They occur most frequently upon the arytenoids, in the interarytenoid space, upon the true cords, and on the epiglottis. The destruction of tissue extends deeply, implicating the submucosa, and in severe cases the perichondrium and cartilages, which undergo more or less extensive necrosis—tuberculous perichondritis and chondritis. The ulcers occasionally extend to the back of the tongue, to the pharynx, to the upper part of the œsophagus, and in severe cases to the pillars of the fauces and the tonsils. Complete erosion of the true cords not infrequently occurs, and the epiglottis is often destroyed throughout the greater part of its extent.

Symptomatology.—The earlier symptoms are those of chronic laryngitis due to other causes. There is slight huskiness, which is at first intermittent, and disappears after resting the voice. It soon becomes continuous, and gives place to a peculiar hoarseness, which in the advanced stages of the disease usually passes into complete aphonia. Cough is tickling, paroxysmal, and unproductive; it has the peculiar quality known as laryngeal, and may be distinguished in the same patient from the mere nervous cough of bronchial irritation. It is at first not distressing, but in cases of advanced ulceration it becomes husky and high-pitched, and is often attended with pain. Spontaneous pain is not a very common symptom. There is often tenderness upon external pressure. Dysphagia is a prominent and most distressing symptom in advanced cases, especially when the epiglottis is involved, the arytenoids are extensively destroyed, or there is ulceration of the pharyngeal wall. In such cases the administration of nourishment is attended with difficulty, the attempt to take food of any kind giving rise to severe pain, urgent paroxysms of cough, and frequently to suffocative attacks. The difficulty in swallowing adds greatly to the sufferings of the patient, and constitutes the most distressing symptom of the terminal stage of this form of tuberculosis. In the earlier stages the laryngoscope reveals the appearances due to chronic laryngeal catarrh. There is, however, greater pallor of the mucous membrane, together with some thickening over the arytenoids. Later the picture is characteristic. The vocal cords are thickened and eroded, and their motility is impaired; the epiglottis and arytenoid are infiltrated, and at various points superficial grayish ulcers with ill-defined borders are seen; finally, deep ulceration, with extensive loss of substance, is superadded.

Diagnosis.—In the earlier stages, and especially in the absence of the evidences of pulmonary tuberculosis, the diagnosis of tuberculous laryngitis cannot be made with certainty. Pallor of the laryngeal mucous membrane, thickening of the arytenoids, general failure of health on the part of the patient, and absence of response to local and constitutional treatment lead to the suspicion of tuberculous disease. This suspicion is confirmed by the appearance of the characteristic ulceration, the evidences of pulmonary tuberculosis, or the detection of tubercle-bacilli in the sputum or the exudate scraped from the floor of the laryngeal ulcer. The differential diagnosis between tuberculosis and syphilis of the larynx is in certain cases attended with some degree of difficulty. In this connection the greater tendency of syphilis to invade the pharynx, the fact that tuberculous ulceration of the larynx is in general progressive and continuously destructive, while syphilitic ulceration frequently shows a disposition to heal at one point while advancing at others, and finally the history of specific inflammatory or ulcerative lesions in other parts of the body in syphilis, will receive due consideration on the part of the practitioner.

Course and Termination.—The course of tuberculous laryngitis is, as a rule, in the highest degree unfavorable. While in the literature of the subject cases of marked amelioration, or even of cure, especially in the primary form, are reported, the disease is so constantly fatal that the instances in which more than a temporary arrest occurs must be regarded as exceptional.

Treatment.—Much can be done, however, by systematic local treatment to retard the progress of the disease. In the earlier stages the measures used with success in chronic catarrhal laryngitis are occasionally followed by benefit. Upon the development of ulceration local cleanliness must be secured by systematic spraying. For this purpose a solution of sodium biborate or a 10-

volume solution of hydrogen dioxide, half strength, may be employed. Direct applications of tannic acid, silver nitrate, or zinc sulphate, in solutions of varying strengths, may be made to the ulcers. The insufflation of iodoform or aristol, with morphine when pain is present, is yet more useful. These applications should be made two or three times a day. Menthol, 20 grains to the ounce of olive oil, is a useful application. I have used methyl-violet in a solution of 20 grains to the ounce with advantage. The treatment by means of a 20 per cent. solution of lactic acid, as suggested by Krause, has yielded favorable results. After the application of cocaine the ulcers are curetted, dried, and then touched with the solution of lactic acid on a pledget of cotton. The strength of the solution is gradually increased. Cocaine, used either in the form of spray or directly applied, yields temporary relief from pain, and often enables the patient to swallow food. In many cases it quickly loses its effect. Tracheotomy has been practised. Bland foods of a semi-fluid consistence are most readily swallowed. Cream, thick gruels, curds, jellies, raw oysters, raw eggs, finely-hashed meats, and ice-cream may often be taken when other articles of diet cannot be swallowed. Rectal alimentation may postpone death by starvation. Wolfenden has suggested that the patient suffering from dysphagia lie face downward on a couch with his head hanging over the lower end, and thus draw into his mouth, by means of rubber tubing, liquid food from a cup upon the floor. Cracked ice may be freely used.

In the severe cases the treatment early becomes simply palliative. When this stage is reached narcotics, especially opium and its derivatives, and cannabis Indica, must be given for effect.

SYPHILITIC LARYNGITIS.

Definition.—Inflammation of the larynx, occurring as a manifestation of syphilis, either hereditary or acquired.

Etiology.—The upper air-passages are especially prone to the ravages of syphilis—a fact doubtless due to their liability to catarrhal inflammation, by which their integrity is impaired and localization of the constitutional virus is determined. The larynx is very frequently involved in syphilitic inflammation.

Morbid Anatomy.—The catarrhal laryngitis of secondary syphilis presents nothing characteristic. Symmetrical superficial ulceration of the true and false cords occurs. Mucous patches, when present elsewhere, confirm the diagnosis, but they are not common in the larynx.

Much more frequent and important are the tertiary lesions. Gummata, multiple or single, develop in the submucous tissues. They may undergo resolution, or, as is much more frequently the case, they break down, giving rise to extensive and deep ulceration, which may involve the cartilages. Sometimes the disease begins as a perichondritis attended with suppuration, and rapidly causing necrosis of the cartilages. In such cases external fistulæ may be formed. In the course of the ulceration erosion of the walls of arterial branches may give rise to free hæmorrhage, or an acute œdema may prove rapidly fatal. The gummata develop most commonly at the base of the epiglottis or in the ventricles. They may attain the size of a nut and occasion serious stenosis of the larynx. The sclerosis which attends their resorption or the cicatrices resulting from the healing of the ulcers often occasion marked deformity of the larynx with progressive stenosis.

The gummatous infiltration of inherited syphilis in either the early or the later form leads to ulceration which tends to extend deeply and involve the cartilages. The healing of such ulcers is also likely to be followed by cicatricial stenosis and deformity.

Symptomatology.—Secondary syphilis of the larynx gives rise to hoarseness and laryngeal irritation. The symptoms of the tertiary lesions are of the most serious character, consisting during the stage of active ulceration of aphonia, cough, pain, dyspnœa, dysphagia, and in the stage of cicatrization of a more or less grave and progressive mechanical obstruction to respiration. The symptoms show themselves in the hereditary disease commonly within the first six months of life; exceptionally, after puberty.

Diagnosis.—The history of the case and of other specific cutaneous inflammatory or ulcerative lesions, or the presence of such lesions or their scars, renders the diagnosis in a majority of the cases a simple matter.

Treatment.—Energetic constitutional treatment is imperative. The local symptoms, as a rule, yield rapidly to mercury and potassium iodide. In neglected cases and in cachectic and broken-down individuals laryngeal ulcerations are difficult to treat. Mercury by inunction or by the hypodermic method frequently succeeds when the ordinary methods of medication fail. Local treatment is of secondary importance. The larynx should be cleansed by detergent sprays, and after cocainization daily application of strong solutions of silver nitrate, gr. c–cxx to the ounce, should be cautiously made to the ulcerated surfaces. Less efficient are insufflations of iodoform or aristol.

The cicatricial stenosis may be held in check by gradual dilatation, with or without division of the net-like bands or adhesions; but this course of treatment is not always satisfactory, and in many of the cases it becomes necessary to perform tracheotomy.

LARYNGISMUS STRIDULUS.

The suffocative paroxysms of the acute laryngitis of childhood (spasmodic croup), and those of the early stages of pseudo-membranous laryngitis (true croup), are largely due to reflex spasm of the glottis. There is, however, a spasm of the glottis in early infancy not in any manner connected with inflammation of the larynx, which, though probably in all instances a symptom, has been described as a separate disease.

Definition.—A neurosis, the prominent symptom of which is spasmodic closure of the glottis, with, in severe attacks, spasm of the diaphragm and other muscles of respiration. The relaxation of the spasm is accompanied by a prolonged, high-pitched, crowing inspiratory sound, from which the affection receives its name.

SYNONYMS.—Spasm of the glottis; Child-crowing; Thymic asthma. Much confusion has arisen from the use of the terms "false," "spurious," and "spasmodic" croup to designate the affection. It is desirable that "croup" should be restricted to those conditions which are associated with inflammation of the larynx. Spasmodic laryngitis, as applied to a purely nervous affection, without any inflammatory condition of the larynx, is a manifest misnomer. The term "thymic asthma" is to be ascribed to the former view, now known to be baseless, that the disease was caused by the enlargement of the thymus gland.

Etiology.—Laryngismus stridulus is a disease of infancy. It occurs almost exclusively before the end of the third year of life. It is more common in boys than in girls. A large proportion of the cases occur in rachitic children. It is also seen in tetany. Children in fairly good health may, however, develop the attack without warning. The predisposition existing, the paroxysm may be excited by a variety of causes, either physical or emotional. Thus, sucking, sudden movements, violent crying, the bath, indigestion, dentition, or a cross word may provoke an attack. The seizures also occur, however, in the absence of such causes—out of sleep, for example—and even more frequently by night than during the day. They often present, especially in older children, a curious appearance of being voluntary, and are sometimes at first regarded as fits of passion or of holding the breath.

Symptomatology.—The attack may be preceded by an occasional catch in the breath or by slight crowing sounds; more commonly it comes on without premonitory symptoms. There is complete arrest of respiration. The chest is fixed; the head thrown back; the face, at first pale, quickly becomes cyanotic; the eyes are wide open and staring. There is often twitching of the facial muscles. In the severer cases there may be opisthotonos, carpo-pedal spasm, or general convulsions. The attack lasts from a few seconds to a minute or more. In very severe cases death takes place during the paroxysm from prolonged stoppage of respiration or from impaction of the epiglottis. As the cyanosis deepens the spasm yields; the air slowly enters the lungs again through the incompletely relaxed glottis, with the characteristic prolonged, high-pitched, crowing sound, and the attack ends in a spell of coughing or crying. The seizures vary greatly in severity and number. After a few repetitions they may cease altogether, or they may come on very frequently, both by day and by night, and recur during a period of months.

Diagnosis.—The absence of fever, hoarseness, and cough in the intervals between the attacks, the suddenness and completeness of the arrest of breathing, the short duration of the paroxysm, the peculiar prolonged crowing inspiration with which it ends, and the associated convulsive phenomena, taken together, form a characteristic clinical picture not to be mistaken for any other malady. In the rare cases in which death occurs in the paroxysm the crowing is absent, and the sudden death from asphyxia may remain unexplained.

Prognosis.—As regards the spasm the outlook is favorable, the fatal cases being few in number. Children who suffer from laryngismus stridulus are, however, as a rule, frail, and a large proportion of them succumb to intercurrent disease.

Treatment.—The child should be raised to the sitting posture. Cold water should be dashed over the face and chest or the surface lightly slapped with a wet towel. Strong ammonia may be held to the nose or the nostrils may be tickled with a feather. Ice applied to the epigastrium, or an ice suppository, may excite inspiratory effort, or a large sponge moistened with hot water held against the throat and chest may cause the spasm to relax. Meanwhile, a hot bath having been prepared, the child should be immersed in it to the shoulders, while cold water is at the same time poured over the head and neck. If the attacks be prolonged, the finger should be pressed into the patient's mouth to discover whether or not the epiglottis may have become incarcerated, and if so to release it. Even after apparent death recovery may follow artificial respiration. If the paroxysms are very frequent and severe, the attendants may be instructed to cautiously administer a whiff or two of chloroform or a few drops of amyl nitrite upon the appearance of signs indicating that an attack is about to occur.

The treatment during the intervals must be directed to the child's general condition. If there be dental irritation and the gums are swollen and livid, they must be freely lanced. The bowels should be opened by calomel or castor oil. Daily warm baths with cold sponging of the chest and back; abundant fresh air; cod-liver oil; iron in the form of the syrup of the iodide; and carefully regulated feeding if the child has been already weaned,—constitute the most useful measures of treatment.

The employment of narcotics and antispasmodics is, as a rule, attended with only partial success in averting the recurrence of the attacks. Among remedies of this group chloral is the most useful.

Attacks in some respects resembling those of laryngismus stridulus are occasionally observed in hysterical adults. The laryngeal crises of locomotor ataxia depend upon spasm of the glottis, which causes severe, sometimes very alarming, dyspnœa and distressing spasmodic nervous cough.

CHRONIC INFANTILE STRIDOR.

I have seen several cases of a rare affection described by Taylor under the above title. It appears shortly after birth, and lasts for a period varying from several months to two years. The chief symptom consists of an almost continuous coarse, low-pitched, inspiratory stridor, which is present both when the child is awake and during sleep. It varies in intensity, being much aggravated by excitement. It sometimes ceases wholly for a few hours. As the disease gradually passes off the stridor occurs only at intervals and when the child is lively or excited. Expiration is usually normal; it may be accompanied by a few coarse mucous râles. Retraction of the thorax scarcely occurs and is slight. In one case only have I encountered faint cyanosis, and in that instance there were during the eighteen minutes of stridulous breathing three transient general convulsions. The case ended in recovery. As a rule, the affection does not seem to interfere with the general health of the child. In a case examined after death Dr. Lees (cited by Taylor) found the epiglottis folded on itself and the ary-epiglottic folds in contact.

Paralysis of the Laryngeal Muscles.

The nerves of the larynx are the superior laryngeal and inferior or recurrent laryngeal branches of the pneumogastric. These are joined by branches of the sympathetic. The superior laryngeal nerves supply the mucous membrane of the upper portion of the larynx, including the epiglottis, as far down as the true cords. They also supply the crico-thyroid muscles, and in fact the thyro-epiglottic and the aryteno-epiglottic muscles, and the arytenoid muscles, which also derive motor filaments from the recurrents. The inferior or recurrent laryngeals curve around the arch of the aorta on the left side and of the subclavian on the right, and ascend between the trachea and œsophagus to supply the laryngeal mucous membrane below the cords, and all the muscles of the larynx except the crico-thyroids. The superior and inferior laryngeal nerves on each side communicate with each other in two places—namely, at the back of the larynx and on the side of the larynx under the ala of the thyroid cartilage. The motor filaments of these branches of the pneumogastric are derived from the spinal accessory nerve.

In paralysis of the laryngeal muscles the lesion may be—

(1) Central, involving the nucleus of the accessory nerve in the medulla. The laryngeal paralyses of this group arise as a result of syphilis affecting the medulla oblongata, acute and chronic bulbar paralysis, multiple sclerosis, and locomotor ataxia. The hysterical paralyses of the larynx must also be regarded as of cerebral origin. Or,

(2) The lesion may affect the fibres of the recurrent laryngeal in the course of the vagus or the accessory nerve. This group includes the cases in which the paralysis is due to pressure by new growths, and there are cases in which the trunk of the nerve is wounded or injured in surgical operations above the point at which the recurrents are given off. Or,

(3) The lesion may directly involve the laryngeal nerves. The majority of the cases of laryngeal paralysis are included in this group. The recurrents are, by reason of their remarkable curves, especially liable to abnormal pressure by new growths, both within the thorax and in the neck. The left, which curves around the aorta, is exposed to greater risk of injury than the right, which passes no lower in the chest than the subclavian. Either may be included in the dense pleural thickening at the apices which occurs in certain forms of pulmonary tuberculosis. Paralysis of the right is in rare instances caused by aneurism of the subclavian artery. The left is likely to be injured by the pressure of an aneurism of the arch of the aorta, a mediastinal tumor, enlargement of the bronchial glands, and in rare cases of a massive pericardial effusion. Both, as they ascend between the trachea and the œsophagus, are occasionally involved in carcinoma of the latter or compressed by enlargement of the thyroid gland.

Paralysis of the recurrents occurs also as a very rare sequel of diphtheria and as a result of chronic alcoholism.

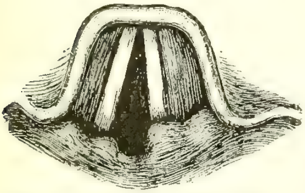
(4) The lesion may be confined to the larynx. The loss of power is purely muscular, and amounts merely to a paresis. This occurs in various diseases, and is due to inflammatory infiltration of the submucous tissues, with altered nutrition of the muscles.

(5) Finally, cases of laryngeal paralysis occur for which no adequate cause can be discovered.

The following are the most important forms of laryngeal paralysis:

1. Complete Paralysis of the Recurrent Nerve.—This condition occurs as the result of lesions dividing or completely destroying the recurrent or its fibres in the vagus, or as a manifestation of neuritis due to diphtheria or other causes, or in consequence of advanced disease in the medulla. It may be unilateral or bilateral. When the paralysis is unilateral the vocal cord on the affected side occupies the median or so-called cadaveric position, and is motionless upon inspiration, expiration, and attempts at phonation (Fig. 30). In phonation the vocal cord and the arytenoid of the sound side pass beyond the median line. The voice is harsh; it easily breaks into a falsetto, and speaking is attended with effort. The cough is likewise harsh and brassy. Dyspnœa is not a symptom. In complete bilateral paralysis—a very rare condition—the cords occupy the median position and are immobile; their edges are slightly concave, as the aperture is sufficiently wide for respiration; dyspnœa is absent except upon exertion. Aphonia is complete and coughing is impossible.

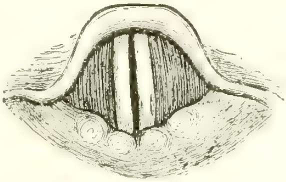
Fig. 30.



Position during inspiration in paralysis of the left vocal cord, or recurrent conduction paralysis (after Ziemssen). Position and immobility of the left vocal cord, as in the cadaver.

2. Bilateral Paralysis of the Abductors.—The posterior crico-arytenoids are involved. This form of laryngeal paralysis may occur as a central affection in the course of bulbar paralysis, multiple sclerosis, and locomotor ataxia. It may be produced by pressure upon both vagi or upon both recurrents. It is also encountered as a rare form of hysterical palsy. Gowers regards it as probable that, in many cases described as hysterical spasm of the glottis, the symptoms are due to paralysis of the abductors. No hypothesis has yet been advanced that is adequate to explain the fact that in pressure-paralysis of the recurrents the filaments supplying the abductors alone are frequently first involved. Abductor paralysis may follow exposure to cold or may arise in the course of a laryngeal catarrh. The cords are approximated as in phonation. The glottis is not opened in inspiration; on the contrary, it acts like a valve, and is narrowed by the pressure of the air to a small slit

Fig. 31.



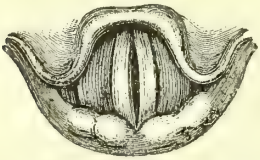
Bilateral complete Posticus Paralysis (paralysis of the crico-arytænoidei postici, dilatation of the glottis) at the moment of inspiration (after Ziemssen).

(Fig. 31). Inspiration is therefore difficult, prolonged, and stridulous, while expiration is unimpeded. Phonation is not affected. The ability to cough remains. This form of laryngeal paralysis is rare, but is attended with the danger of sudden suffocation. If the symptoms are progressive and the dyspnœa constant, tracheotomy becomes necessary and the tube must be constantly worn.

3. UNILATERAL ABDUCTOR PARALYSIS.—One cord only may be affected in pressure-paralysis involving the recurrent of one side. Aneurism of the arch of the aorta, exerting pressure upon the left nerve, is by far the most common cause of this condition. The right nerve is especially liable to be involved in pleural thickening and retraction of the apex of the lung in the course of pulmonary tuberculosis. The vocal cord on the affected side remains fixed in the middle line during inspiration. The voice is sometimes unaffected; more commonly it is slightly harsh or rough. Dyspnœa and stridor are not often present. The movements of the other cord are normal.

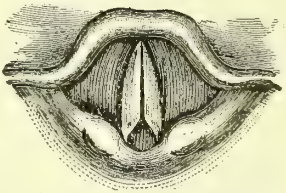
4. ADDUCTOR PARALYSIS.—In the more common forms of adductor paralysis the lateral crico-arytenoids, the arytenoid, and the thyro-arytenoids are implicated. It occurs as the result of exposure to cold or from over-use of the voice, and is very often the cause of loss of voice in catarrhal laryngitis; it is the usual form of paralysis in hysterical aphonia. The laryngoscope reveals the normal position and movement of the cords in respiration, but their total failure to approximate on attempts at phonation. There is neither stridor nor dyspnœa; ability to cough is not affected, but aphonia is complete. Adductor paralysis may be partial. It is commonly bilateral, but in exceptional cases unilateral. In bilateral paresis of the thyro-arytenoids, the glottis does not close completely on phonation, the margins of the cords being separated by an oval space (Fig. 32). If one cord only is affected, its margin remains concave. In paralysis of the arytenoid, which scarcely occurs alone, the vocal cords are brought together in their anterior extent, but the failure of the arytenoid cartilages to approximate leaves a narrow triangular opening at the inter-arytenoid space (Fig. 33).

FIG. 32.



Paralysis of both Thyreo-arytænoidei Interni, resulting from acute laryngitis (after Ziemssen). Position during phonation.

FIG. 33.



Paralysis of the Arytænoideus in Acute Laryngitis (after Ziemssen). The posterior portion of the glottis remains open during phonation.

DISEASES OF THE BRONCHI.

BY JAMES C. WILSON.

CATARRHAL BRONCHITIS.

Definition.—Catarrhal inflammation of a portion or the whole of the mucous membrane of the bronchial tubes, except the ultimate subdivisions, the bronchioles.

The last are not involved in the inflammatory process without the implication of the corresponding vesicular structure. The pathological condition is then properly described by the term "broncho-pneumonia."

The mucous membrane of the trachea frequently shares to some extent in catarrhal inflammation of the larynx, and is usually also affected in bronchitis of the larger tubes. As catarrhal tracheitis can scarcely be said to occur as an independent affection, and as its symptoms are usually subordinate to those of the laryngitis or the bronchitis with which it is associated, it seems neither in accordance with clinical experience nor necessary to systematic teaching to describe it as a separate disease.

The term "capillary bronchitis" has been used to designate a very common and serious condition which arises in the course of attacks of acute primary bronchitis of severe type alike in young children and in the aged, and is of frequent occurrence in the secondary forms of bronchitis. This condition, of which the symptoms and physical signs, as well as the post-mortem appearances, are very well defined and characteristic, is now known to be due to the extension of the inflammatory process, not only to the smaller tubes, but also to the bronchioles and to the alveolar structure of the lung. It is fully described under the heading Broncho-pneumonia.

Besides this use to designate an affection implicating the lung-tissue as well as the bronchi, the term "capillary bronchitis" has been applied to pulmonary atelectasis, a condition which may, under certain circumstances, occur in feeble children without previous bronchitis; it has also been employed to describe a condition in which the physical signs or the rational symptoms justify the suspicion that broncho-pneumonia is about to develop, or even to describe the early stage of broncho-pneumonia. Yet, again, there are those who would restrict the term "capillary bronchitis" to an inflammation of the finest subdivisions of the bronchial tubes, but not involving the bronchioles; that is, as the last point to which the catarrhal process may extend without involving the vesicular structure—a condition which manifests itself neither by definite clinical phenomena nor by constant post-mortem lesions.

The various uses of the term "capillary bronchitis" have given rise to much difficulty and confusion in the minds of students. Neither clinicians nor pathologists recognize "capillary bronchitis" as an independent affection. The term is not only useless; it is also positively misleading, and should therefore be suffered to pass into disuse.

SYNONYM.—Bronchial catarrh.

Catarrhal bronchitis may be—(1) acute, or (2) chronic.

1. ACUTE BRONCHITIS.

Etiology.—Acute catarrhal inflammation of the mucous membrane lining the trachea and larger bronchial tubes is a common disease. It is frequently caused by exposure to cold or damp or by these two influences combined. It is not necessary that such exposure should be general; on the contrary, an attack of bronchitis is more likely to follow the chilling of a portion of the surface, such as takes place from wetting the feet or sitting on damp ground, than the ordinary exposure resulting from the vicissitudes of the weather. The inhalation of cold, damp air may directly affect the upper air-passages, and thus induce bronchial catarrh. The affection is not infrequently due to the extension of an ordinary coryza. The inhalation of air charged with smoke or dust, or containing the fumes of certain chemicals, as sulphurous acid, chlorine, or bromine, may give rise to acute bronchial catarrh. When the susceptibility is great, and especially in children, acute bronchitis, like coryza, is mildly contagious; hence the occasional prevalence of house-epidemics. Such restricted outbreaks and the more extended local epidemics which prevail in the absence of influenza can often be traced to local causes, as extreme and sudden changes in the weather, fogs, dampness, and other meteorologic influences. The acute forms arising from the foregoing causes are known as primary.

Secondary or associated bronchitis is encountered with great frequency in the course of acute and chronic diseases. A distinction must be made between those forms which constitute a manifestation of the primary disease and those which arise as complications. To the former belong the bronchitis of influenza, of whooping-cough, of the early stages of typhoid fever, and that which accompanies the exanthemata, as measles and variola. The bronchial catarrh in these diseases is due to the localization of the infection in the mucous membrane of the respiratory tract, and is therefore symptomatic. To the second of these two groups we properly refer the cases of acute bronchial catarrh which arise in connection with severe diseases of the mouth and throat, as diphtheria when the bronchitis is not the result of direct extension, tuberculosis and syphilis of the larynx, erysipelas, quinsy, glossitis, and the like; those which prove so dangerous in the later course of grave acute diseases, as typhoid, typhus, and scarlet fevers, or in the advanced stages of degenerative diseases of the nervous system, as multiple sclerosis, locomotor ataxia, and bulbar paralysis; and, finally, the bronchial catarrh which frequently develops as a result of wounds of and surgical operations upon the upper air-passages.

In the latter group the bronchial catarrh is due to secondary local infection caused by retained or inspired secretions and foreign substances, and must be regarded as a complication.

Among predisposing influences climate is of great importance. Bronchitis is especially a disease of cold, damp, and changeable climates and of the variable weather of early spring and late autumn. The liability to "catch cold" is greatly increased by fatigue and by the condition of the skin induced by violent exertion. Acute bronchial catarrh is a disease of all periods of life, but the very young and the aged are especially prone both to the attack and to its more severe manifestations. The period of the first dentition is attended with a notable liability to catarrhal diseases of the respiratory as well as of the intestinal tract.

The mode of life is of importance as a predisposing influence. Bad sanitary arrangements, including defective drainage, deficient ventilation, overcrowding, extremes of temperature, improper clothing, and insufficient food, enhance the disposition to attacks of acute bronchitis. Those who follow out-of-door avocations are less liable than those who lead sedentary lives. Finally, many individuals possess a peculiar constitutional predisposition to bronchial catarrh.

Morbid Anatomy.—The lesions are bilateral. They affect the larger, medium-sized, and in severe cases the smaller tubes. When the bronchitis is restricted in extent, the tubes passing to the lower lobes are usually implicated. The mucous membrane is reddened, congested, and swollen. Its glands are enlarged. Its surface is bathed in catarrhal secretion, consisting at first of altered mucus containing degenerated epithelium; later, of muco-pus with an abundance of epithelial cells and leucocytes. In severe and protracted cases the submucosa is swollen, œdematous, and infiltrated with leucocytes.

Symptomatology.—The constitutional symptoms are usually those of "taking cold." After exposure, which may or may not have attracted the patient's attention, there is chilliness, usually slight, scarcely ever amounting to a chill, accompanied by depression and lassitude, and followed by pains in the back and limbs. There are now also headache and flushing. Sensations of chilliness and discomfort are excited by the slightest draught of air, by changes of clothes, or even by movement. Many of the cases are subacute rather than acute. In these there is little fever, the temperature not exceeding 100° F.; in the severer forms it may reach from 101°–103° F. A higher temperature will arouse the suspicion of an extension of the inflammation to the vesicular structure.

The chest-symptoms consist of a feeling of rawness and soreness, with oppression, cough, expectoration, and, if there be associated laryngitis, hoarseness. Pain is often absent. When present it is usually of moderate intensity. It is much aggravated for the moment by paroxysms of cough. It is usually substernal or referred to the intercostal muscles and the insertion of the diaphragm. A sense of oppression is common in the early stages. It is usually transient, and in ordinary cases of bronchitis of the larger and medium-sized tubes does not amount to dyspnœa. When the finer tubes are implicated, or when the catarrhal secretion is excessive, there is often marked shortness of breath. Cough is a constant symptom: in many cases it is the first to attract the patient's attention. In the early stage of the attack it is paroxysmal, rough, hoarse, and accompanied by pain. It is then dry or attended with scanty and difficult expectoration. In the course of a few days the sputum becomes abundant, muco-purulent in character, and is expectorated without effort. With this change the patient experiences decided relief, especially as regards the oppression and pain. The cough likewise loses its paroxysmal character and ceases to rack and distress him. The expectoration is at the outset viscid, light-colored, and scanty. Occasionally, especially in the severer cases, it may be streaked with blood. Later it becomes less tenacious and more opaque, as well as more abundant, and is seen microscopically to consist chiefly of pus-corpuscles with large cells containing the so-called myelin droplets of Virchow and carbon particles. In infants the expectoration is usually swallowed.

Physical Signs.—Upon laryngoscopic examination, if the mucous membrane of the trachea be implicated, it will under favorable conditions be seen to be reddened and the seat of increased secretion.

Upon inspection the chest presents no morbid physical signs except when there is active fever or the inflammation has extended to the finer tubes: under these circumstances there is acceleration of the respiratory movements and slight prolongation of expiration. This increase in respiratory frequency is associated with an increase in the pulse-rate, and is proportionately more common in children than in adults. Percussion in cases of uncomplicated bronchial catarrh yields negative results. Auscultation in many of the milder cases, affecting the larger tubes only, discovers no abnormal physical signs—a fact that the student must not overlook. But commonly in the early stages there are heard a few scattered sonorous with many sibilant râles. These are not constant, but appear and disappear, and are much affected by cough. Later the changes in the character of the secretion are manifested by corresponding changes in the râles, which become moist, bubbling, and of varying coarseness—large, medium-sized, and small mucous râles. When the finer tubes are involved, subcrepitant râles may be detected. In the later stages, especially when the secretion is abundant and fluid and the chest-walls are thin, as is the case in children, a rhonchal or bronchial fremitus is detected on palpation. A similar fremitus occasionally accompanies the dry, sonorous râles of the early period of the disease.

The course of primary acute bronchitis in healthy adults is favorable. In a few days the fever subsides, the more troublesome symptoms disappear, and a gradually decreasing cough with diminishing expectoration is left. In the course of two or three weeks this also ceases, and recovery takes place without sequels. In the previously debilitated, the very young, and the aged there is great danger that the process may extend to the finer tubes, and thence to the air-vesicles. In the debilitated and the aged this tendency is favored by the difficulty in expelling the secretion which accumulates in the finer tubes, especially at the bases; in infants and young children it is further increased by the relative looseness and vascularity of the bronchial mucosa, and a more active proliferation of its epithelium as a result of irritation. The tendency to the development of broncho-pneumonia is even more marked in the secondary forms of bronchial catarrh, especially those which occur in the course of measles, whooping-cough, influenza, and the severe cases of other infectious diseases. Extension to the finer tubes is followed by narrowing of their lumen from swelling of the mucous membrane, plugging by secretions, areas of collapse, and by the development of broncho-pneumonia. It is marked by further rise in temperature, great increase in the rate of the respiration and pulse, and often by faint cyanosis. The physical signs marking the change consist of abundant subcrepitant râles at the bases, posteriorly and elsewhere, circumscribed areas of impaired percussion-resonance, and feeble or distinct bronchial breathing. In cases in which there is stupor, as in the so-called typhoid condition, this extension usually takes place without change in the symptoms, and may be overlooked in the absence of systematic physical examination of the chest as a matter of routine.

Diagnosis.—The diagnosis of acute bronchitis is rarely attended with difficulty. The mode of onset, the oppression, the paroxysmal cough, the râles, and the moderate fever are usually sufficient to lead to a direct diagnosis. If the onset be abrupt and the fever high, croupous pneumonia may be suspected —a suspicion speedily set at rest by the absence of dulness, crepitus, pleural pain, and rusty sputum. It is important to differentiate the bronchial catarrhs which are primary from those which are symptomatic or secondary, as in influenza and whooping-cough. The localized catarrhs of pulmonary tuberculosis are to be recognized by their unilateral character, the accompanying dulness, and the presence in the sputum of tubercle-bacilli or elastic fibres. In acute miliary tuberculosis the diagnosis is not always at first possible. The gravity of the illness, its progressive character, the irregularity and occasional intensity of the fever, the disproportion between the dyspnœa and the physical signs, constitute a clinical picture not seen in ordinary acute bronchial catarrh.

Prognosis.—In healthy adults the prognosis is favorable. Neglected cases may develop the chronic form—a tendency which is observed in those who are the subjects of advanced heart-disease or kidney-disease, and in the gouty; also in those whose occupations habitually subject them to the inhalation of a vitiated atmosphere or to frequent, sudden, or prolonged exposure to damp and cold. In infancy and old age, and in those enfeebled by previous illness or vicious living, and in the secondary forms, acute bronchial catarrh becomes, chiefly by reason of the danger of broncho-pneumonia, a very serious and frequently fatal disease.

Treatment.—The subacute cases of bronchitis in healthy persons tend to early recovery and require very little treatment. A Dover's powder, reinforced by a hot mustard foot-bath and a free draught of hot lemonade at bedtime, will often suffice. A portion of whiskey or gin may be added to the lemonade. Other diaphoretics, as the warm bath, sweet spirit of nitre, liquor ammonii acetatis, or Warburg's tincture, may be preferred. Those who are in the habit of taking Turkish baths frequently seek to "break a cold" by that means —a practice attended with very great risk of adding to the depression already existing, and increasing the congestion of the bronchial mucous membrane in the exposure incurred in leaving the bath-house, and therefore not to be recommended. At the onset, nothing is so useful as free diaphoresis. The next morning a mild saline may be given with advantage, and followed in the course of a few hours by ten grains of quinine. More severe cases require careful management, especially at the extremes of life and in delicate persons. The patient must keep his room; if there be fever, his bed. If the cough be distressing, and especially if there be oppression or dyspnœa, the air of the room must be kept moist by steam. The direct inhalation of steam, alone or impregnated with benzoin or terebene, also affords relief to the cough. Attempts to treat bronchitis by inhalation have not yielded satisfactory results. The chest-pains are relieved by the occasional application of a mustard plaster, a turpentine stupe, or, especially if there be difficulty in breathing, by dry cupping. Cut cups are not, as a rule, indicated in bronchitis even of severe type.

The diet should be simple and restricted. In the early stages it should consist chiefly of hot liquids—tea and toast, gruels, thin porridge, soups, and the like. A mixture of equal parts of milk and Vichy water, either hot or cold, is useful and acceptable. After the secretions are re-established, and in particular if the expectoration be very free, solid food must be given and the quantity of liquid taken somewhat curtailed.

The medicinal treatment is symptomatic. Fever may be controlled by the administration of small doses of tincture of aconite in neutral mixture, or of antipyrin, or of phenacetin. Cough is alleviated by small doses of Dover's powder or Tully's powder at intervals of three or four hours, or morphine dissolved in cherry-laurel water. Codeine may be used for this purpose in cases in which opium is not well borne. Opium and its derivatives are to be used with extreme caution in the bronchial catarrh of infants and of aged persons, especially when the secretion is abundant; but, properly employed, they are of great value in relieving the distressing dry cough of the early stage, at which period they appear in many cases to exercise, in addition to relief of the cough, a beneficial influence upon the general condition of the patient and the course of the disease.

During the dry stage, and especially when this is prolonged, it is customary to administer expectorants, as ipecacuanha and antimony, either of which may be given in the form of the wine, or the wines may be given together in spirit of Mindererus, with the addition of the sweet spirit of nitre. Ipecacuanha may also be given in the form of the syrup, or tartar emetic alone in minute doses. A convenient method of prescribing the latter is to dissolve one grain in a tumbler of cold water and give a teaspoonful every hour. An attack of bronchial catarrh may sometimes be aborted by the administration of

$\frac{1}{12}$ grain of tartar emetic every twenty minutes until vomiting is induced. Apomorphine in doses of $\frac{1}{20}$–$\frac{1}{10}$ grain every two hours is a soothing expectorant, and speedily brings about copious secretion.

In many cases the loosening of the cough and an abundant expectoration indicate the beginning of convalescence. No further medication is then required. It is customary, however, at this stage of the disease, to give ammonium chloride, senega, and squill, separately or in various combinations. These are difficult to take, often interfere with the appetite, and in many cases are of questionable advantage. They should, therefore, in no case be prescribed except in response to clear indications. If the catarrh tends to become chronic or the secretion remains too free, turpentine, terebene, terpin hydrate, tar-water or tar-syrup, cubebs, or oil of sandal may be employed, according to the requirements of individual cases. Under these circumstances iron and arsenic in anæmic subjects, and a temporary change of air, especially to a region of pines with a dry, sandy soil, will prove useful. In feeble persons strychnine, digitalis, and caffeine, and the judicious use of alcohol, will aid in averting the complication of broncho-pneumonia; and these measures are especially indicated in all forms of secondary bronchitis.

In children the treatment should begin with a calomel purge. A single dose of from one to three grains may be given, or quarter-grain or half-grain doses may be repeated at short intervals until the desired effect is produced. Mild purgation may be advantageously secured from time to time during the course of the attack, by means of either calomel or rhubarb and soda. The child should be kept in bed and carefully guarded from draughts. The temperature of the room should be maintained uniformly at about 70° F. When there is troublesome paroxysmal cough, oppression, or when the bronchitis is associated with laryngitis, the air should be kept moist by means of steam from a croup-kettle or other suitable contrivance.

The diet must be digestible and nutritious. During the acute stage it should consist of liquids only. In young infants the addition of peptonoids to the ordinary food is often of benefit, and the use of alcohol is always indicated in feeble and debilitated patients and whenever the symptoms and signs point to involvement of the smaller tubes. Mild counter-irritation by means of camphorated oil or turpentine, or preferably amber oil, morning and evening when the child's clothing is changed, or more frequently, is of use. Poultices and jackets of cotton and oiled silk are inconvenient and unnecessary. The fever yields to minute doses of aconite; even more satisfactorily to antipyrin. For the cough one selects paregoric as safest, codeine as most efficient, apomorphine as the promptest. These may be administered in water flavored with syrup of tolu or Aubergier's syrup of lactucarium; the addition of minute doses of chloroform enhances the sedative effects of such cough-mixtures. Small doses of Dover's powder, from one-quarter to one grain at intervals, serve a like purpose. Extreme caution, I repeat, is to be observed in the use of these drugs, and it is never right to prescribe morphine to young children. Small doses of ipecacuanha in the form of the wine or syrup, the syrup of garlic, and such demulcents as slippery elm and flaxseed tea, are administered with a view of hastening the loosening of the secretion. As expectorants children bear squill, senega, and the carbonate and chloride of ammonium better than adults. Tartar emetic is a dangerous remedy in childhood, and should not be employed. Quinine, useful during the convalescence and in cutting short mild attack in adults, is without effect upon the symptoms and course of the disease in children.

When the secretion is abundant and not freely expectorated, and in particular if dyspnœa supervene and the lips and finger-nails are livid, an emetic must be administered. For this purpose ipecacuanha or the yellow subsulphate of mercury is to be preferred. The prompt evacuation of the stomach brought about by apomorphine is less likely to be attended by expulsion of bronchial secretion than the more prolonged and general effects caused by other emetics; moreover, the administration of apomorphine in emetic doses is attended with the danger of causing collapse. The warm bath, with the application of cold water to the head and neck while the child is in the bath, constitutes a most useful treatment of acute bronchial catarrh in children. It may be repeated three or four times in twenty-four hours, and is usually followed by quiet sleep, diminished cough, deeper breathing, and other signs of general improvement.

The **prophylaxis** consists in the avoidance of the etiological factors heretofore indicated; the use of proper clothing, including flannels; systematic bathing, such as is suited to the individual; daily cold sponging and frictions of the neck and chest; and respiratory gymnastics. In every case of severe illness involving the danger of secondary bronchitis the nose and mouth must be systematically treated by detergent sprays or washes; care must be taken that in the administration of food the patient's attention is sufficiently aroused; he must, if possible, be made to repeat several times a day a series of deep inspirations; his position must be frequently changed; and deep breathing be excited by cold sponging or even cold affusion of the chest.

2. CHRONIC BRONCHITIS.

Etiology.—Chronic bronchial catarrh may follow a neglected attack of acute bronchitis. Much more frequently it gradually develops after repeated attacks of the subacute form, the unfavorable conditions which excite subacute bronchitis giving rise, by their frequent repetition or permanence, to chronic bronchitis. In by far the greater number of instances, however, it occurs in association with other chronic diseases, as of the lungs, heart, and kidneys, and with gout. It is a disease of the degenerative period of life. Hence it is common in the aged. It is rare in the young, and, occurring in children, is usually associated with rickets or follows whooping-cough, measles, or broncho-pneumonia. Sex has, of itself, little determining influence. Occupation is, however, of importance; hence chronic bronchitis is more common in men than women. Climate and season constitute predisposing influences of great moment, the chronic like the acute disease being more frequent in cold, damp, and changeable climates, and more troublesome in cold and variable weather. In elderly persons the symptoms often abate in summer, to recur with regularity upon the return of winter; hence the term "winter cough."

Morbid Anatomy.—The lesions vary greatly both in character and extent. The bronchial epithelium is altered, often absent throughout extensive areas; the mucous membranes are thinned; and there is atrophy of the mucous glands; or the mucous membrane may be irregularly thickened and granular, with points of ulceration. In cases of long standing atrophic changes take place in the muscular tissues and the cartilages, and cylindrical dilatations of the bronchial tubes (bronchiectasis) are found. Secondary changes in the vesicular structure give rise to emphysema. Deficient oxygenation and mechanical interference with the pulmonary circulation result in dilatation of the right heart.

Symptomatology.—The primary symptoms of chronic bronchitis consist in disturbances of respiration, cough, and expectoration. In mild cases and in the beginning of cases that afterward become severe, other symptoms are absent, and the general health of the patient is not greatly impaired. After a time, however, emphysema, bronchiectasis, dilatation of the right heart, and visceral congestions gradually develop, and give rise to secondary symptoms of great importance. The health is then seriously and progressively impaired. The fever and constitutional disturbance of the acute form are wanting. Pain is absent. Dyspnœa, especially upon exertion, may be present in cases of uncomplicated chronic bronchitis if the morbid process be extensive and attended with abundant secretion; as a rule, it is due to the secondary changes in the heart or lungs. In certain cases asthmatic attacks occur. The cough has no constant character. It is likely to be more troublesome at night or in the morning than during the day. It is often paroxysmal and distressing, especially in cases in which the expectoration is scanty. It is much influenced by season and climate, being less troublesome in summer and in warm, dry climates than in winter and in cold or damp weather. Under these circumstances the general condition of the patient often undergoes a corresponding improvement. The sputum is very variable in character and quantity. In a group of cases, the so-called dry catarrh is unattended with expectoration, or this is at most occa-

FIG. 34.

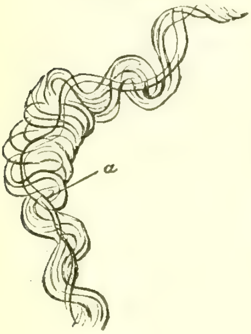


FIG. 35.

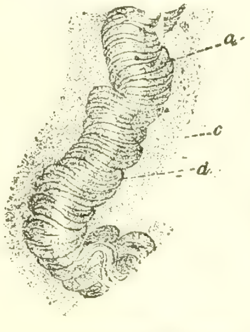
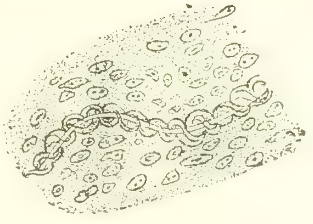


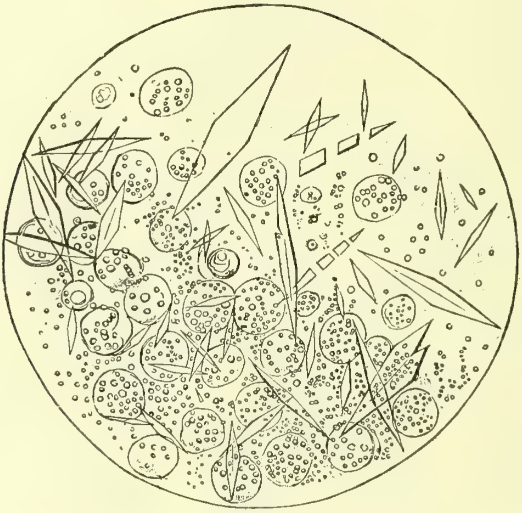
FIG. 36.



Curschmann's Spirals.

sional and scanty. In another group, the sputum is of moderate amount, thin, glairy, and translucent. It is commonly copious and muco-purulent or purulent, varying from a thin, slightly frothy fluid to a thick secretion of yellowish or greenish-yellow color, with all the characteristics of pus. In general, the sputum is without characteristic odor; at times it has, however, a faint, mawkish smell, and occasionally, especially when associated with bronchiectasis, an intense penetrating fœtor. In individual cases the odor undergoes marked change from time to time. The minute masses of blood-clot occasionally seen in the sputum of chronic bronchitis are without special significance. Microscopically, the sputum is found to contain pus-corpuscles, fatty epithelium, myelin droplets, and a variety of micro-organisms. In cases complicated with asthma the sputum during and just after the paroxysms contains Curschmann's spirals and Charcot-Leyden crystals.

FIG. 37.



Charcot-Leyden Crystals. (After Riegel.)

Physical Signs.—In cases of uncomplicated chronic bronchitis the signs on inspection are negative. At most there is slight prolongation of the movement of expiration. Mensuration yields negative results. On palpation, if there be much secretion, a rhonchal fremitus may be felt. On percussion the resonance is normal or vesiculo-tympanitic (hyper-resonant), or in the presence of abundant secretion, accumulated for the moment at the bases, the resonance is slightly impaired. Upon auscultation the normal vesicular murmur may be heard, but more commonly the murmur is broncho-vesicular; it is often obscured by râles of all kinds, dry, sonorous, or sibilant, moist or bubbling, both coarse and fine, while at the bases posteriorly occasional subcrepitant and crepitant râles are heard. If emphysema be present, as is usual in well-developed cases of chronic bronchitis, the chest is distended and the respiratory movement limited.

Varieties.—In practice several different forms of chronic bronchitis may be distinguished. These clinical varieties are not always distinctly separated; they occasionally merge into one another in the course of the disease.

1. The greater number of the cases are of mild character and slowly progressive. They constitute the "winter cough" of old persons, and are usually associated with emphysema or cardiac disease. This form of chronic bronchial catarrh is common in the gouty. The cough is variable, often paroxysmal, more troublesome at night than during the day. In the morning on rising it is often severe and prolonged, and does not cease until the secretion accumulated during the night is expelled. The sputum varies in character and amount, its peculiarities being largely determined by the extent and nature of the lesions.

2. A mild form of chronic bronchitis of different kind is occasionally seen. It occurs chiefly, but by no means exclusively, in women, and begins at an earlier period of life. It is not very amenable to treatment, and lasts indefinitely, without serious impairment of the general health. There is morning cough, with occasional paroxysms during the day, often provoked by effort or excitement, and not influenced by the weather. The sputum is not copious, and may consist of small masses of glairy mucus, dislodged with difficulty, or of a somewhat more abundant muco-pus. Physical examination yields negative results. There are no râles; emphysema does not develop; and the heart is usually unaffected. Osler has described these cases. I have seen them in members of gouty families.

3. BRONCHORRHŒA is the term used to designate chronic bronchitis attended with excessive secretion. The expectoration is either clear and transparent or opaque and purulent; in either case it is thin, but in the latter it frequently contains tough greenish masses; in exceptional cases it is thick and uniform. It usually contains little air, and on standing separates into layers, the purulent portion accumulating at the bottom of the vessel, the serum above this, and a thin layer of frothy mucus collecting at the top. There are abundant râles, and when the secretion accumulates, slight impairment of resonance at the bases posteriorly can be demonstrated. In cases characterized by thin, clear sputum the paroxysms of cough are often prolonged and distressing, and there is difficulty in breathing. The expectoration is copious, not rarely reaching four or five pints in the course of twenty-four hours. This condition may last indefinitely, without serious implication of the vesicular structure of the lungs, and is not incompatible with a fair degree of general health. The cases in which the sputum is purulent are those in which the lesions of the bronchial tubes are advanced, and are associated with beginning bronchiectasis. The sputum in these cases does not ordinarily exceed one or two pints in twenty-four hours.

4. FŒTID BRONCHITIS.—Fœtid expectoration occurs in the course of bronchiectasis, in connection with tuberculous cavities in the lung with retained and decomposing contents, in connection with abscess and gangrene of the lungs, and in empyema with bronchial fistula. It is met with in rare cases

of chronic bronchial catarrh in the absence of these conditions—primary fœtid bronchitis. The expectoration is abundant, usually thin, and of a grayish-green color. It has a most disagreeable sweetish, putrid odor. On standing it separates into an upper frothy layer, a middle dirty-green stratum of thin, sero-mucous fluid, and a sediment composed chiefly of pus, in which may be seen with the unaided eye small whitish-gray masses—Dittrich's plugs. These masses consist of broken-down pus-corpuscles, detritus, bacteria of various kinds, and acicular crystals of fatty acids arranged in bundles. Tufts of fungi which color blue with iodine and potassium iodide solution are also present in the sputum, and Lumniczer has separated a fungus which, cultivated on agar-agar, emitted the odor characteristic of fœtid bronchitis. Chemical examination of the sputum discloses the presence of leucin, tyrosin, volatile fatty acids, sulphuretted hydrogen, and other products characteristic of putrefactive decomposition. The breath of the patient has the same penetrating, foul odor as the sputum. When in the course of a chronic bronchial catarrh putrefactive changes in the bronchial secretion take place, there is usually a coincident aggravation of the symptoms. Repeated chills occur, followed by high fever, with increased cough and pain in the chest.

Fœtid bronchitis may lead to ulceration and dilatation of the bronchial tubes and to pneumonia; it is frequently followed by abscess or gangrene of the lung and purulent pleurisy, and in a small porportion of cases metastatic abscess of the brain has resulted. In many cases the acute symptoms which mark the onset of the putrid change subside; the fœtor continues; and the case resumes the course of one of ordinary chronic bronchitis, often with much gastric disturbance and pains in the joints, due to the absorption of septic matters.

5. Dry Catarrh.—There is little expectoration or none at all. When present the sputum is tough and dislodged with difficulty. The cough is paroxysmal and distressing. There is a tendency to asthmatic seizures. On auscultation sonorous and sibilant râles are heard, but mucous râles are absent. This form of chronic bronchitis is almost always associated with emphysema. It is common in old persons, and is not very amenable to treatment. It is the *catarrhe sec* of Laennec.

The Course of the Disease.—Chronic bronchitis has a very protracted course. Under favorable circumstances, and especially in good weather, marked and prolonged remissions take place; the cough and other symptoms become less troublesome; and the patient's condition is comparatively comfortable. Upon the recurrence of cold weather, however, there is an exacerbation of the disease; the symptoms return with full force; and for a period the affection makes progress. Such remissions and exacerbations are frequent, but in general the condition of the patient grows gradually worse. Complete recovery is very rare indeed.

Complications.—After a time, the length of which varies greatly in different cases, and which is much influenced by the care which the patient takes of himself, emphysema develops and there is dilatation of the bronchial tubes.

So constant is the association of these conditions with chronic bronchitis that they may almost be regarded as part of the disease. The condition of the bronchial mucous membrane favors the implantation of the tubercle-bacillus; hence chronic tuberculous pleurisy and chronic pulmonary tuberculosis, especially the form known as fibroid phthisis, are not infrequently developed in the course of a chronic bronchial catarrh. Secondary emphysema gives rise to gradual hypertrophy and dilatation of the right ventricle, which in turn are followed by transference of blood-pressure from the arterial to the venous circulation, with visceral congestions, breathlessness, œdema of the extremities and grave progressive impairment of the general functions.

Diagnosis.—The diagnosis of chronic bronchitis is usually made without difficulty. It rests upon the history of the disease, the symptoms, and the physical examination. It is of the utmost importance to determine whether the disease is primary or secondary. The frequency with which chronic bronchial catarrh is associated with—nay, dependent upon—disease of the heart and arteries, of the kidneys, or upon gout, demands the closest investigation of the general condition of the patient and of the condition of the organs of circulation and excretion in particular.

In cases characterized by fœtid expectoration it is important to determine whether the disease is essentially a chronic putrid catarrh or whether we have to deal with abscess or gangrene of the lung, with a cavity formed either by dilated bronchi or the breaking down of a caseous tuberculous mass, the contents of which have undergone putrid change, or with an empyema discharging by way of a bronchial fistula. The differential diagnosis between primary fœtid bronchitis and certain forms of abscess and gangrene of the lung is attended with some difficulty. The existence of areas of circumscribed dulness and the presence in the sputum of shreds of lung-tissue with elastic fibres, crystals of hæmatoidin, amorphous blood-pigment, and crystals of cholesterin are in favor of pulmonary abscess. In gangrene shreds of lung-tissue are likewise present in the sputum, but elastic fibres are not always found separately—a fact due, according to von Jaksch, to the presence of a ferment which acts like the pancreatic juice, and which probably causes the solution of elastic tissue. Bronchiectasis and vomicæ are to be recognized in repeated examination by their physical signs, and the one-sided flatness; bulging intercostal spaces, and change of contour of the chest in empyema render the diagnosis of this condition a comparatively simple matter. The presence of a fistulous communication with the lung will already have converted the empyema into a pyo-pneumothorax, the signs of which are usually obvious.

Prognosis.—The prognosis as regards ultimate recovery is not favorable; as regards length of days and a fair degree of general health, it is moderately good. From the latter point of view it is much affected by the circumstances of the patient and his ability to avail himself of the advantages accruing from a well-regulated manner of living and change of climate. The danger while the disease is in its early advance is to be looked for in the development of morbid changes in the lungs and heart. The prognosis in the secondary forms of chronic bronchitis depends upon the prognosis of the primary disease and the extent to which the latter is capable of successful management.

Treatment.—The importance of the careful management of a case of acute bronchitis until convalescence is complete, and the avoidance of those conditions, whether of occupation or of climate, by which repeated attacks of subacute bronchitis are caused, cannot be overestimated, especially in those past middle life who manifest a predisposition to bronchial catarrh. When the affection has assumed a chronic form, the injury is permanent, and its tendency in most cases is to become gradually but surely progressive. Measures of prophylaxis are therefore of paramount importance. In what these measures consist has already been set forth, in the main, in the discussion of the treatment of acute bronchitis. An additional means of prevention is found in change of climate. Early removal to a warm, equable climate may avert the onset of the threatened disease. The climate of the Riviera, Capri, Algiers, Florida, and Southern California renders these localities suitable winter resorts for those predisposed to bronchial catarrh.

The less fortunate persons who are unable to avail themselves of the advantages of change of climate must be impressed with the necessity of avoiding those conditions by which bronchial irritation is excited. To this end a change of occupation is frequently essential. The neck and chest are to be habitually sponged with cold water on rising, after which the circulation of the skin is to be excited by brisk friction. Flannels must be worn throughout the year, their weight being cautiously varied to suit the season. The outer clothing should be of moderate weight and not too close-fitting. It is scarcely necessary in this connection to inveigh against the uselessness of the heavy absurdities called chest-protectors. Especial care must be taken in regard to the protection of the feet from dampness and cold. The dwelling should be maintained at an even temperature of about 68° F., and should be thoroughly ventilated. The patient, especially in bad weather, must undergo the hardship of avoiding crowded places of amusement, which are often unduly heated and without proper ventilation. Draughts are dangerous; but the open air in dry, clear weather, even though the cold be decided, is to be much sought. It is among the advantages of health-resorts that those who frequent them live largely in the open air in an atmosphere free from dust.

These measures of precaution are not less useful in the management of the declared disease. They are more efficacious than drugs. Food must be digestible and moderate in quantity. The sparing use of wine or whiskey, well diluted, may be permitted elderly persons. All excess must be avoided. The alkaline mineral waters, as Seltzer, Vichy, etc., are useful adjuncts to the general treatment. Inhalations are much employed in the treatment of chronic bronchitis. When the secretions are scanty, steam, a 10 per cent. solution of sodium chloride, or a weak solution of sodium bicarbonate, is to be preferred; when they are abundant, turpentine by inhalation is useful. The most convenient method is to place a teaspoonful of turpentine in a small pitcher of boiling water and inhale the steam as it arises; it may also be inhaled by means of a flask through the cork of which a tube passes below the level of a quantity of turpentine floated upon water, while a second tube, bent at an angle for the mouth-piece, merely penetrates the stopper, so that, at each inspiration, air containing turpentine vapor is inhaled. Pure terebene may also be used in the same manner, the dose for a single inhalation being from five to ten minims. Prolonged inhalations of these substances may be practised by means of oro-nasal respirators; a few drops of turpentine or five drops of pure terebene being used from time to time. An excellent mixture for this purpose consists of equal parts of terebene, carbolic acid, and spirit of chloroform: the dose for a single inhalation is 10 drops. This mixture is especially useful in cases of fœtid bronchitis, in which a spray of carbolic-acid solution of 2 or 3 per cent. strength, or of thymol, 1 : 1000, is also of advantage. The patient should use a spitcup containing a disinfectant and deodorizing solution, and a spray of carbolic acid, 10 per cent., should be occasionally used in the room. While inhalations are useful in the treatment of certain forms of chronic bronchitis, they cannot be wholly depended upon.

It is necessary to mention the treatment by means of special pneumatic apparatus (Waldenburg, Cohen, and others) which enables the patient to inhale and expire into air of different densities. This use of rarefied and compressed air is of some little service in the emphysema with which chronic bronchitis is commonly associated, but it has not fully realized the claims made for it by its advocates, nor has it been generally adopted in the practice of physicians.

After measures of hygiene our chief reliance is placed upon internal medication. In dry bronchitis expectorants are employed. Ipecac; apomorphine, because of its soothing effect upon the troublesome cough; terpin hydrate in doses of from 2 to 4 grains every two or three hours—are among the more useful. The stimulating expectorants, squill, senega, and ammoniac, are to be given with caution and for short periods. If good effects follow, their administration may after a time be resumed and kept up in short courses frequently interrupted. Potassium iodide, in doses of from 5 to 15 grains three times a day, appears in certain cases to exert a curative influence. It should be tried in every case of primary bronchitis which tends to become chronic. All depressing drugs are contraindicated. When the secretion is abundant, ammonium chloride in full doses, from 10 to 15 grains, every two hours, is very useful. The balsams are employed with good results in these cases. Turpentine may be given in emulsion or in capsule, 10 minims three or four times a day; or its isomer terebene in capsule, 5 minims two or three times a day; or terpin hydrate, which in larger doses, 6 grains three or four times a day, checks expectoration. The last should be given before food. In advanced cases, with much muco-purulent or purulent expectoration, oil of sandal, cubeb, and copaiba are valuable, and the first often promptly yields gratifying results. The compound tincture of benzoin, the balsam of Peru, and the balsam of tolu, formerly much used in the treatment of chronic bronchial catarrh, are not efficient remedies in ordinary doses, and have fallen into disuse in the therapeutics of this disease. Tolu is, however, on account of its flavor, agreeable to many persons, and is still used in the preparation of various cough mixtures. The fluid extract of cheken has been extolled as a remedy for chronic bronchitis; that of grindelia is certainly of service in cases complicated with asthma. The preparations of tar are especially valuable in the winter cough of elderly persons. Myrtol has been used with benefit in the treatment of primary fœtid bronchitis. The dose of myrtol is from 2 to 5 minims in capsule, repeated several times in the course of the day. It reduces the expectoration in amount, diminishes the fœtor, and doubtless, in consequence of these effects rather than of any constitutional influence, exercises a salutary impression upon the general condition of the patient. Other remedies which do good in fœtid bronchitis are oil of sandal and terpin hydrate.

Narcotics, and in particular opium and its derivatives, possess the dangerous power of controlling cough and affording temporary relief in the more distressing forms of this disease. They rapidly lose their effect, however, and in the increasing doses which become necessary they derange important functions and additionally impair the health. The greatest danger which attends their use is that of the formation of the opium-habit—a calamity immeasurably greater than that from which the patient already suffers. The administration of these drugs is to be avoided in this as in other chronic diseases until the last stages, when their judicious use will often greatly mitigate the suffering from which death affords the only escape. Their early employment begets a tolerance which not only robs them of their usefulness when this is in greatest need, but also adds new sufferings to those which belong to the disease.

The complications demand special treatment. Potassium iodide and arsenic are useful for emphysema; digitalis and strychnine for the affections of the right side of the heart which gradually develop.

In the secondary forms of chronic bronchitis treatment must be directed to the primary disease. Success in the management of mural or valvular disease of the heart, renal insufficiency, whether functional or organic, and gout, will usually be followed by marked relief of the chronic bronchial symptoms which arise as secondary manifestations of these affections.

BRONCHIECTASIS.

Definition.—Dilatation of the bronchial tubes.

Bronchiectasis is not a substantive disease. It is an anatomical lesion which occurs in connection with primary and secondary affections of the bronchi. In cases of well-marked dilatation of the bronchi a new group of symptoms and signs is sometimes added to those of the pre-existing malady. Two forms of bronchiectasis are met with—the cylindrical and the saccular. These may be found together in the same case.

Etiology.—Inflammation of the bronchial mucous membrane, extending to the submucous structures, and leading to atrophic changes in the muscular and fibrous elements and the cartilages, is the essential cause of bronchial dilatation in a large proportion of the cases. The walls become unable to withstand the pressure of the air in severe paroxysms of cough. The accumulation of secretion tends by its weight to distend the weakened walls, and further impairs their elasticity and resistance by soakage. This group includes the bronchiectasis of chronic bronchitis and emphysema, the dilatation of the smaller tubes in the broncho-pneumonia of children, that which results from the presence of foreign bodies in the bronchi and from compression by an aneurism or other tumor. In another group of cases the contraction of the fibrous framework of the lung in pulmonary cirrhosis and fibroid phthisis leads to dilatation of the bronchi by traction upon their walls from the outside. To this category are to be referred those instances of bronchiectasis which are met with in cases of ancient pleurisy, cirrhosis of the lung, whether due to pneumonokoniosis, fibroid phthisis, or interstitial pneumonia, and those which occur in cases of chronic pulmonary tuberculosis, in which interstitial changes play an important part.

Very rare instances have been reported in which diffuse bronchiectasis affecting one lung has occurred as a congenital defect or anomaly.

Morbid Anatomy.—Cylindrical bronchiectasis consists in an enlargement of the calibre of bronchial tubes, more or less uniform and extending for some distance. It involves the medium-sized, less frequently the finer, tubes of one or more lobes of the lung. By reason of the uniform enlargement, the anatomical condition is not always recognized, being sometimes mistaken for a larger tube. This error is not likely to occur if the branch from which the twig is given off is noticed to be of smaller diameter. This form of bronchiectasis is more frequently due to those morbid conditions in which weakening of the walls and paroxysmal increase of internal pressure are associated. The process by which it comes to pass is a gradual one.

Saccular bronchiectasis consists in the formation of oval or irregularly spherical dilatations involving a limited portion of a bronchial tube. These dilatations, which vary in size from a pea to a small orange, are usually multiple. Single saccular bronchiectatic cavities resembling vomicæ are occasionally encountered in cases of chronic bronchitis and emphysema. The wall of the bronchial tube is thinned ; its constituent tissues are atrophied in greater or less degree, and the cavity often presents the appearance of the interior of a simple cyst without contents. Sometimes the wall of the dilated bronchus is traversed by bands of connective tissue, and not infrequently there is ulceration. Occasionally a small bronchus may be seen to open into the sac. Saccular bronchiectasis may present the appearance of a series of bead-like dilatations at short irregular intervals in the course of the smaller bronchi. Congenital bronchiectasis is of this form. It occurs as a series of small saccular dilatations opening one into the other, and affects the whole of one lung, the surrounding tissue being densely indurated. The walls are thin and lined by a smooth, glistening membrane, not ulcerated except in the more dependent parts of the lung. In rare instances only do we encounter a single saccular bronchiectasis surrounded by non-indurated lung-tissue. Such a condition occurs in cases of bronchitis and emphysema, and is due to atrophic changes in the bronchial wall and pressure within the bronchus. As a rule, to which there are few exceptions, saccular bronchiectasis, both congenital and acquired, occurs in a lung that has undergone cirrhotic or fibroid changes. The cavities are immediately surrounded by an indurated and contracting connective tissue, bands of which extend throughout the affected lung. Pleural adhesions are uniformly present, and the theory of Corrigan, that the fixation of the lung at its periphery and its subsequent contraction cause dilatation of the bronchi, by traction upon the walls from without, has been generally received. The mucous membrane is greatly altered in all cases. In the smaller cylindrical dilatations it may be infiltrated and thickened, so that with stretched walls the lumen of the tubules is little enlarged ; in the larger saccular bronchiectasis, with smooth and glistening walls, the columnar is replaced by pavement epithelium.

Symptomatology.—Moderate dilatation of the bronchial tubes does not, as a rule, give rise to special symptoms or physical signs. Nothing is added to the morbid phenomena of the case of chronic bronchitis, emphysema, or contraction of the lung in which it occurs. It cannot therefore be recognized during life. In large saccular bronchiectasis, however, a new group of symptoms gradually arises. The changed anatomical peculiarities of the bronchi give rise to special clinical phenomena. These relate to the cough and expectoration. The peculiarity of the cough in bronchiectasis consists in its occurring in prolonged paroxysms, at intervals of several, often many, hours, and its being accompanied by excessive and frequently foul-smelling expectoration. The sputum collects in the cavity of the dilated bronchus, without giving rise to disturbance, but upon reaching a given amount or upon change of posture it wells over to a neighboring tube, which responds to the irritation of its presence by cough, which continues until the accumulation is discharged. It is evident that the spell of coughing will frequently take place on arising in the morning. It may, however, occur in the course of the day and at irregular intervals. The expectorated matter, gray or brownish in color, of fluid consistence and sour odor, is frequently fœtid. Allowed to settle, it separates into three layers—an upper of brownish froth, the middle of watery mucoid fluid, and a thick sediment of granular matter and cells. The sediment, examined microscopically, is seen to consist chiefly of pus-corpuscles, among which are numerous crystals of the fatty acids arranged in bundles. When there is ulceration of the wall of the bronchial tube crystals of hæmatoidin and elastic fibres may be present. More or less hæmoptysis may occur under these circumstances. This is, however, rare. In uncomplicated cases tubercle-bacilli are not present in the sputum or in the expectorated blood. Not rarely the sputum is extremely and persistently fœtid, with the other characteristics of that of fœtid bronchitis. The nummular sputa common in cases of pulmonary tuberculosis do not usually occur in bronchiectasis.

Physical Signs.—Extensive saccular bronchiectasis presents the signs of pulmonary cavities. These signs vary from time to time, according to the amount of accumulated secretion, and are more commonly localized at the base of the lung. The affection is unilateral. Cavernous and amphoric signs are usually distinct. The associated chronic pleurisy and interstitial pneumonia may give rise to retraction of the chest-wall, with permanent recession of the intercostal spaces—a sign not always well marked. Impaired resonance, bronchial respiration, and increased vocal fremitus are met with over the adjacent surface.

Course.—Bronchiectasis is, as a rule, the outcome of chronic disease, and does not terminate in recovery. It is, however, not incompatible with a certain degree of general health and an active life, in this respect resembling some of the forms of bronchitis and interstitial pneumonia with which it is associated. It adds, however, new dangers to these conditions, and ultimately gives rise to severe symptoms, either in consequence of dilatation and hypertrophy of the right ventricle or by reason of progressive emphysema, the development of tuberculosis, or pulmonary gangrene.

Diagnosis.—Cylindrical bronchiectasis in its ordinary form cannot be diagnosticated during life. The large saccular form may be recognized by the character of the cough and expectoration, the physical signs of one or more cavities, and the association of those conditions of the lung in which this form of bronchiectasis occurs. The absence of tubercle-bacilli from the sputum, the greater frequency of saccular bronchiectasis at the base than at the apex, the chronic course of the disease, and the absence of implication of the apex of the opposite lung, serve to simplify the diagnosis between this lesion and the ulcerative cavities of pulmonary tuberculosis—vomicæ. As the physical condition of a circumscribed empyema communicating by means of a pleural fistula with a bronchus is not unlike that of a saccular bronchiectasis near the periphery of the lung, so the differential diagnosis between these conditions is difficult to a high degree. The history of the case is of importance: in bronchiectasis the patient has suffered either from chronic bronchitis or interstitial pneumonia of long standing, while in circumscribed empyema there is usually an account of unilateral trouble of acute onset, without previous impairment of health; or after ill-health, without definite symptoms other than oppression and dyspnœa upon effort, the patient suddenly begins to expectorate at intervals large amounts of pus.

Treatment.—The medicinal treatment of bronchiectasis is that of the causal disease in which it originates. When the dilatation has reached a degree recognizable clinically, it constitutes a permanent lesion. The paroxysmal cough is productive and beneficial; narcotics are therefore contraindicated. Experience has shown that stimulant expectorants are useless. Balsamic drugs are often administered with benefit. Among the more useful of this group are terpin hydrate in full doses, terebene, and turpentine. The latter two are efficient when there is much fœtor, in which case santal oil and myrtol given internally are also valuable, and carbolic acid in from 1 to 3 per cent. solution, or thymol 1 : 1000 by inhalation. In certain cases the fœtor is persistent and offensive to a high degree. It thus constitutes one of the most distressing symptoms of the case. When inhalations and internal medication are alike useless in relieving it, intrathoracic injection of disinfectants may be successfully practised; or in selected cases, in which the cavity is near the surface, it may be freely opened and drained.

FIBRINOUS BRONCHITIS.

Definition.—A rare affection of the bronchial mucous membrane, characterized by the formation of branching fibrinous casts of portions of the bronchial-tube system, which are expelled at intervals in violent paroxysms of cough and dyspnœa.

SYNONYMS.—Pseudo-membranous bronchitis; Croupous bronchitis; Plastic bronchitis.

Fibrinous or croupous formations take place in the trachea and bronchi in several diseases. Small fibrinous casts of the bronchioles may occasionally be found in the sputum of croupous pneumonia. These result from the coagulation of the exudate in the finer tubules, and may be discovered by the unaided eye if the sputum is agitated in water. Similar but more extensive casts are sometimes discovered in the sputum of cases of pulmonary tuberculosis. In cases of hæmoptysis more or less extensive moulds of the bronchial tubes are often formed by the coagulating blood. These casts may be detected by washing the expectorated clots in a basin of water, and are in certain cases of conclusive diagnostic importance in determining the source of blood discharged from the mouth. Finally, in cases of diphtheria of the larynx fibrinous pseudo-membranous formations frequently invade the trachea and bronchi. These membraniform exudates form continuous linings of the tubes, and not infrequently extend from the larynx to the smaller tubules. Occasionally they are not continuous, but, after extending some distance from the larynx into the trachea or larger bronchi, they cease, to reappear in the finer tubules and bronchioles as tubular linings or as solid plugs.

True fibrinous bronchitis is a substantive affection different from all of these conditions, and is not to be confounded with them. It may be acute or chronic.

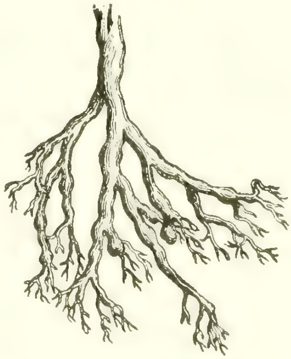
Etiology.—The causation of fibrinous bronchitis remains as yet in complete obscurity. It has been assumed from analogy that in this as in other croupous inflammations of mucous membranes destruction of the epithelium must first occur; but nothing is actually known as to the essential nature of the process or the agency by which it is brought about. It is more common in males than in females. It occurs at almost all periods of life, but the liability appears to be greatest in the third and fourth decades. I have found no record of a case under the age of three. The attacks occur with somewhat greater frequency in the spring of the year than at other seasons. Instances have been noted in which several members of the same household, and again in which a number of individuals in the same locality, have been attacked at short intervals by fibrinous bronchitis—a fact which warrants the view that the disease may be due, under these circumstances, to local endemic infection. It is not contagious. In some instances the attacks have appeared during the menstrual periods; they have occurred in patients suffering from affections of the skin, as herpes and pemphigus, and during the course of acute infectious diseases, as typhoid fever. A number of cases have been recorded in which the patients have suffered from disease of the heart. It is probable that the association with these conditions has been accidental rather than causal.

Less infrequent and more definite is the connection of fibrinous bronchitis with pulmonary tuberculosis. Such is the frequency of this relationship that it has led to the separation of the cases into two groups—one including the essential or primary cases of fibrinous bronchitis, occurring in persons previously healthy, and the other the symptomatic or secondary forms which arise in the course of other diseases, especially in the course of pulmonary tuberculosis; and it has been assumed that the etiological relations of these two forms of the disease are not the same. If, however, fibrinous bronchitis be due to a specific infection, the two forms probably arise from the same or similar exciting causes, the lesions of chronic bronchitis and tuberculosis of the lungs especially acting as predisposing influences.

Morbid Anatomy.—The morbid anatomy remains obscure, owing chiefly to the fact that the disease is rare and the opportunities for study have been few. A loss of epithelium has been noted in the affected bronchi. Fatal cases have in most instances presented the lesions of antecedent or associated disease, such as chronic pleurisy, tuberculosis, or pneumonia. The difficulties surrounding the pathology of the disease relate chiefly to the denseness of the casts, which in this respect differ from ordinary croupous formations; their restriction to limited branches of the bronchial tree; and their re-formation at varying regular or irregular intervals through long periods of time.

Symptomatology.—The acute cases are even much more rare than the chronic. The disease begins suddenly, with fever, and in some cases with chills or chilly sensations. There is severe paroxysmal cough, dyspnœa, and pain in the chest. Early hæmoptysis sometimes occurs. The attack presents the phenomena of an ordinary acute bronchitis of unusual severity, and not until the expulsion, usually after the lapse of some days, of the characteristic fibrinous casts of the bronchial tubules does its true nature become evident. These casts are of a yellowish-white color, and in consistency tough and elastic. They are usually expectorated rolled up in a mass of mucus or muco-pus, which is not infrequently stained with blood or admixed with blood-clot. When the sputum is placed in water the casts often spontaneously unfold or they are readily unravelled. The cast then appears as a complete mould of a bronchus of the second or third order, with its branches. The casts vary in size and in length, the main stem sometimes exceeding one-third of an inch in diameter, and the whole branching cast reaching three inches or more in length. It has

Fig. 38.



Fibrinous Bronchial Cast.

frequently been noted that in the same patient the casts have, in repeated attacks, been precisely alike in size and form, suggesting their formation in the same part of the bronchial tree. They are in places irregularly swollen. In transverse section they usually present a free lumen; exceptionally they appear as solid plugs. In either case they are seen to be made up of concentric laminæ of coagulated fibrin, which for the most part retains its fibrillated appearance, but in some places has undergone hyaline degeneration. In its network are imbedded leucocytes and red corpuscles, and in the lumen of the smaller twigs are often found alveolar epithelium and carbon particles. Leyden's crystals and the spirals of Curschmann are also frequently found in the sputum of fibrinous bronchitis.

These casts are expectorated after very urgent paroxysms of cough, attended with dyspnœa and suffocative symptoms, and not infrequently with blood-spitting. Upon the expulsion of the cast the urgent symptoms at once abate. While the attack lasts the paroxysm may recur at intervals of a day or two. In the intervening periods the symptoms do not greatly differ from those of a simple acute bronchitis.

The **physical signs** are those of acute bronchitis, and, as a rule, present nothing characteristic. In theory the plugging of a bronchus of the second or third order with its ramifications would give rise to suppression of the respiratory murmur throughout an area of considerable size, and to diminished respiratory movement in the corresponding region; in practice, however, these changes are not always to be discovered upon careful physical exploration, even in cases that, owing to repeated attacks, have afforded excellent opportunities for the investigation.

The **duration** of the acute cases is from a few days to three or four weeks. In cases that run a favorable course the fever subsides, the cough becomes less frequent, the casts are no longer expectorated, and the attack terminates in complete recovery. It is uncommon for the acute to pass into the chronic form. In severe cases the symptoms rapidly augment in intensity, and death may occur in a paroxysm of suffocation.

The chronic form of fibrinous bronchitis develops with catarrhal symptoms, but usually without fever. The cough becomes urgent and paroxysmal; the sputa are blood-stained, or—and this is not uncommon—there may be profuse blood-spitting. At length, after a spell of unusually distressing cough, with symptoms of suffocation and cyanosis, the patient expels a tough mass, which upon examination proves to be a bronchial cast. Relief follows. Even the catarrh may for a time pass away. More commonly it persists, and in the course of a few days or a week, or after a longer interval, the attack is repeated. The intervals are variable. In some instances they are prolonged, and may extend to a year or more. Sometimes they are regular, the attack recurring at definite periods. The disease may last for many months, or even for years. In some cases the patients maintain a fair degree of general health, notwithstanding the severity of the attacks; in others chronic bronchitis of severe type and pulmonary tuberculosis supervene.

Diagnosis.—The diagnosis rests upon the finding of the casts in the sputum. It is needless here to point out the importance of a careful search for them in every case of bronchitis attended with violent paroxysms of cough and dyspnœa. Certain cases reported as fibrinous bronchitis have been diphtheritic. In diphtheria the presence of an exudate upon the tonsils, palate, or pharynx is of diagnostic value, and the expectorated intralaryngeal new-formation is shreddy, membraniform, and irregular, not often exhibiting the complete orderly branching of a bronchus and its subdivisions, and never showing the admirable concentric lamination characteristic of the casts of fibrinous bronchitis. A bacteriological study which reveals in the questionable membrane the presence of Klebs-Loeffler bacilli would settle its diphtheritic nature.

Prognosis.—In the acute cases a guarded prognosis is rendered necessary by the fact that the mortality is high. In the chronic cases the prognosis is favorable as regards prolonged life, but very indefinite as regards recovery, as they are likely to continue for months or years, and as in the course of a considerable proportion, other chronic or acute disease of the lungs develops.

Treatment.—In the acute form the treatment is that of simple acute bronchitis. The inhalation of steam or the use of alkaline sprays, especially of lime-water, favors the separation of the casts. Pilocarpine, which frequently increases the bronchial secretion, has been recommended for this purpose on theoretical grounds. In severe paroxysms the cautious administration of ether affords relief and aids in the loosening of the casts. Their expectoration is assisted by the use of emetics of ipecacuanha or turpeth mineral. Potassium iodide and mercurials have been given in the chronic cases until their constitutional effects were produced, without modifying the course of the disease or preventing the recurrence of the attacks. In fact, no treatment capable of preventing recurrence is known, and in the intervals of the attacks the cases must be managed in accordance with the rules laid down for the treatment of chronic bronchial catarrh in its ordinary forms.

DISEASES OF THE PLEURA.

BY JAMES C. WILSON.

PLEURISY.

Definition.—Inflammation of one or both pleural membranes.

SYNONYM.—Pleuritis.

Pleurisy presents all the varieties common to inflammation of serous sacs. The cases may therefore be grouped according to several principles of classification: Etiologically, as simple or idiopathic, tuberculous, cancerous, infectious, or septic, and so on; or, again, as primary and secondary; anatomically, as circumscribed and diffuse; or, in accordance with the character of the exudate, as dry, plastic, or fibrinous on the one hand, and pleurisy with effusion on the other, the latter including sero-fibrinous, purulent (or empyema), and hæmorrhagic pleurisy. Finally, upon the basis of their clinical course the cases may be divided into: I, acute, and II, chronic; and in the present state of our knowledge of the etiology of the serous inflammations, this general arrangement is probably the most convenient for purposes of description.

I. ACUTE PLEURISY.

Two anatomical forms require separate consideration: 1, dry, plastic, or fibrinous (*pleuritis sicca*); and 2, pleurisy with effusion (*pleuritis humida*), including *a*, sero-fibrinous pleurisy; *b*, purulent pleurisy or empyema; and *c*, hæmorrhagic pleurisy.

1. DRY, PLASTIC, OR FIBRINOUS PLEURISY.—**Etiology.**—This form may be primary or secondary. The primary form is that in which pleurisy occurs as an independent affection in persons previously healthy. It is much less common than was formerly supposed. A large proportion of cases of dry pleurisy are now known to be secondary to tuberculosis or other morbid process previously latent or undetected. Nevertheless, cases occasionally occur in individuals who both before and after the attack are in good health. In such cases the disease is usually attributed to cold and exposure; sometimes it follows a contusion of the chest. The secondary form of fibrinous pleurisy is an almost constant accompaniment of acute and chronic pulmonary affections which involve the periphery of the lung. The visceral and, secondarily, the corresponding costal pleura become inflamed. Pleurisy is a secondary process in all cases of croupous pneumonia, except certain rare central pneumonias, and in many cases of broncho-pneumonia. It occurs also in conjunction with

hæmorrhagic infarct, with abscess and gangrene, and with cancerous and other tumors of the lung. It is an accompaniment of all forms of pulmonary tubercle, except acute diffuse miliary tuberculosis, and in many of the cases the symptoms of pleurisy are the earliest evidences of the tuberculous process.

Morbid Anatomy.—The lesions of plastic pleurisy are usually circumscribed. The inflamed serous membrane is deeply injected and shows at various points minute ecchymoses. It has lost its smooth surface and lustrous appearance, and is dry and dull—changes due to the presence of a thin layer of coagulated fibrinous exudate. When the exudate is more abundant, it appears as a distinct granular investment of lymph, which may present a shaggy appearance from the friction of the two pleural surfaces, or may be thick and made up of several distinct strata. The exudate is of a yellowish-gray or reddish-gray color. It consists chiefly of fibrin, with leucocytes, red blood-corpuscles, and serum, in slight but varying proportions, the last, however, in this form of pleurisy being increased little or not at all and undergoing rapid resorption. The presence of this fibrinous exudate upon opposing surfaces of the inflamed pleura accounts for the more important of the rational symptoms and physical signs, and directly results in the formation of permanent adhesions, which, in the course of time, become dense and firm. Such adhesions are frequently found upon post-mortem examination in cases in which there are no pulmonary lesions, and in which no history of chest-disease has been elicited, the symptoms of fibrinous pleurisy in the past having been overlooked or forgotten.

Symptomatology.—The constitutional symptoms of fibrinous pleurisy are not usually severe. In the secondary forms they are very often merged with those of the primary disease. The onset, though frequently abrupt, is not often attended by the chill and sudden access of fever observed in pneumonia. There is pain in the side, fever of moderate grade, a dry cough. Upon auscultation friction-sounds are heard. The fever rapidly subsides, the cough disappears, the pain gradually loses its intensity, and in the course of a few days convalescence is complete. The friction-sound often outlasts the other manifestations of the disease, and can be detected upon deep breathing for a day or two after the other symptoms have disappeared.

2. PLEURISY WITH EFFUSION.—It very frequently happens that the inflammatory process does not end with the formation of a fibrinous exudate containing in its meshes an insignificant proportion of serum, leucocytes, and blood, but goes on to the production of a variable amount of fluid exudation, constituting a pleural effusion. The constituent elements of the exudate remain the same, but their proportions are greatly changed. When the serum is largely increased, the condition is known as serous or, as fibrin is always present, sero-fibrinous pleurisy. When together with much serum there is an excess of cellular elements and the fluid is sero-pus or pus, the condition is known as purulent pleurisy or empyema ; and in the comparatively rare cases in which the effusion consists largely of blood the condition is designated hæmorrhagic pleurisy.

a. Sero-fibrinous Pleurisy.—**Etiology.**—The causes of this form of pleural inflammation are the same as those of plastic pleurisy, the differences in the exudate being determined by differences in the intensity of the process, by constitutional peculiarities on the part of the patient, by the nature of other morbid processes with which the pleurisy is associated, and doubtless by other factors of which we have no knowledge. The affection may be primary or secondary. The primary cases often rapidly follow exposure to cold or wet or an injury to the chest. The part played by various pathogenic micro-organisms in the causation of pleurisy has not yet been by any means definitely settled. There is a tendency to ascribe an increasing proportion of apparently primary cases to microbic origin. That a very large number of such cases of pleural effusion are tuberculous is made evident by the presence of recent tuberculous lesions upon examination when death has occurred shortly after the pleurisy has shown itself, or by the subsequent rapid development of tuberculosis. That many of the secondary cases are tuberculous is clearly shown by familiar clinical and pathological experience. Fibrinous pleurisy is not only present to a marked extent in almost all cases of pulmonary tuberculosis, but in an important group of the cases it even constitutes the earliest evidence of the tuberculous process. Insidious sero-fibrinous pleurisy is very frequently the conspicuous condition in tuberculous disease in which the lung-symptoms play a secondary but characteristic part. Furthermore, a limited tuberculous focus is occasionally found in the lungs, which, without causing marked pulmonary symptoms, may have been the source of pleural infection; and, finally, the acute pleurisy which arises as a secondary process in various chronic visceral diseases is very often tuberculous. In the present state of knowledge concerning the course of tubercle in serous membranes, as shown especially in tuberculous peritonitis, in which recovery may take place spontaneously and after cœliotomy, we cannot assign very great importance to the fact that a large proportion of the cases of acute pleurisy terminate in recovery, as opposed to the view that the affection is frequently tuberculous. Sero-fibrinous pleurisy may arise as a secondary process in the course of many acute and chronic diseases of the lungs. It may also occur in association with certain forms of inflammation of other serous membranes, as pericarditis and peritonitis, and arises as an occasional complication in typhoid fever, influenza, and other acute infectious diseases, acute rheumatism, gout, various forms of chronic nephritis, in chronic affections of the liver, as cirrhosis, and in cancer.

While pleurisy is a disease of all periods of life, it occurs more frequently in adults than in children. It is more common in men than in women, and slightly more frequent in male than in female children. More cases arise in the cold and changeable weather of the winter and spring than at other seasons of the year.

Morbid Anatomy.—The pleural surfaces are covered with a coagulated fibrinous exudate, which varies in thickness and arrangement in different cases. It may consist of a thin, rather smooth, uniform coating, with few shreds of lymph in the effusion; or it may be made up of thick, superimposed, soft layers, the surface of which is shaggy, with abundant flocculent masses in the fluid effusion; or in rare cases it assumes a loose, irregularly honeycombed arrangement, in the meshes and interstices of which the fluid is collected. It usually involves the interlobar surfaces, which are thus rendered adherent. The effusion collects in a single mass, which, unless prevented by previously existing adhesions, gravitates to the dependent portion of the pleural cavity. Exceptionally it is divided by the septa formed by the stretching of recent adhesions into several distinct collections, not communicating with each other—multilocular pleural effusions. It varies greatly in amount. A quantity less than ten ounces (300 cc.) does not in the adult give rise to appreciable physical signs. It may exceed four litres (eight pints). In a child a year old three ounces (100 cc.) will give rise to distinct and extended dulness on percussion. The fluid exudate is of a pale-yellow or yellowish-green color, and, as it contains a variable number of cellular elements, slightly turbid. In chemical composition it resembles the serum of the blood. It is rich in albumin, of a specific gravity varying from 1015 to 1023, and often, upon standing, undergoes spontaneous coagulation. A trace of sugar may be found. Upon microscopical examination leucocytes, cells derived from the pleural endothelium, red blood-corpuscles, and fibrin-threads are found, and in old effusions crystals of cholesterin and occasionally uric acid.

The changes in adjacent organs are chiefly mechanical. They become progressively striking and important as the effusion increases in amount. The presence of a small quantity of fluid in the pleural cavity permits the lower lobe of the lung to retract; as the effusion increases the relaxation of the whole lung results in slight displacement of the mediastinum toward the opposite side, in consequence of the traction of the sound lung. The pulmonary tissue immediately overlying the effusion becomes atelectatic. Further increase gives rise to positive pressure upon the lung and the adjacent structures, and in massive effusions reaching to the clavicle the lung is compressed into an elongated, flattened mass, airless as regards its vesicular structure and almost bloodless, which occupies a position close to the vertebral column. The mediastinum and diaphragm yield to the pressure of the accumulating fluid. The heart is displaced toward the opposite side. In cases of effusion into the right pleural sac the liver occupies a position lower than normal; in cases of left-sided effusion there is downward displacement of the stomach and transverse colon, and to some extent, especially in children, of the spleen. Marked depression of the diaphragm tends somewhat to counteract the effect of direct lateral pressure upon the mediastinum, and modifies the displacement of the heart. Osler has shown that with the most extensive left-sided effusion the apex of the heart is not rotated, and does not pass to the right of the median line; that the relative positions of the apex and the base are usually maintained; and that the right chambers of the heart lie to the front as in health. He concludes that the displacement is rather a definite dislocation of the mediastinum, with the pericardium, to the right, than any special twisting of the heart itself. In no instance, in a number of post-mortem examinations made for the purpose of determining these points, did he discover the abrupt twist of the inferior vena cava described by Bartels, Fraentzel, and others, and which has been invoked as the cause of sudden death in large left-sided effusions. Modifications of the foregoing displacements, both of the lung and adjacent organs, are effected by firm pleural adhesions and by the presence of tumors of the thoracic or abdominal cavity or other local infradiaphragmatic morbid conditions, such as ascites or excessive tympanites.

Symptomatology.—Acute sero-fibrinous pleurisy very often begins with slight shivering, which may be repeated during several successive days. There is rarely a single, severe initial chill, with high fever. This latter form of onset suggests the concurrence of a circumscribed pneumonia, the other symptoms of which are not well defined. A large proportion of the cases develop insidiously. Especially do those secondary effusions which occur as complications, and frequently as the terminal event, in the course of nephritis, cancer, and other chronic diseases, often come on without preceding symptoms. It is not, as is sometimes asserted, that the symptoms of pleurisy are sometimes overlooked in the prominence of those which characterize the primary disease, but that the onset of the pleural inflammation is unattended by symptoms, and the disease remains latent until a considerable effusion is discovered upon physical examination or upon the post-mortem table. Pleurisy with effusion, occurring as a secondary process in the course of other acute affections, is commonly accompanied by early chest-pain, rise in temperature, and pressure-symptoms, and is not likely to escape observation, especially if the methods of physical examination be systematically employed.

The febrile movement is not typical. The rise is neither so abrupt nor so great as in pneumonia. In the course of three or four days the temperature reaches 102° or 103° F.; in cases of sero-fibrinous effusion it rarely exceeds 104° F. In a week or ten days, during which the morning remissions are trifling and the evening range remains about the same, the fever may subside, or it may persist for three or four weeks. Apyrexia may be complete, while the signs of effusion remain unchanged. From this we infer that the fever is a symptom of pleural inflammation, and not necessarily of the presence of a serous exudate, even when large, in the pleural sac. Persistence of fever, especially when with an evening rise to 104° F. or beyond there are morning remissions to normal or below, in some instances justifies the suspicion of active tuberculosis of the lungs, but more commonly that we have to do with an effusion that has undergone purulent changes or has been purulent from the outset. It is certainly true that persistent fever of a hectic type is the rule in purulent pleurisies, but undue importance may be ascribed to this fact. There are occasional exceptions. In a limited proportion of cases fever of this type accompanies effusions which upon aspiration prove to be sero-fibrinous; in yet fewer empyema is present without fever. Such exceptions to the general rule occur alike in adults and in children, but are more frequent in early life.

The surface temperature on the affected side is frequently between one and two degrees higher than on the sound side. A marked and persistent difference is much less common with sero-fibrinous pleurisy than with empyema.

Pain in the side is a common and characteristic symptom. In the latent form it may, however, be insignificant or entirely absent. It is usually an early symptom, but it may not appear until a few hours, or even a day or two, after the beginning of the disease. It is commonly sharp and lancinating, and is described as a "stitch in the side;" exceptionally it is tearing or dragging. The pain of pleurisy is very often limited to a small area below the nipple or in the axillary region. It may be more diffuse, and is sometimes referred to the infraclavicular or infrascapular region, or it may be retrosternal. In rare cases, especially in diaphragmatic pleurisy, no pain is felt in the chest, but it is referred to the hypochondriac region or to the epigastrium or to the lumbar region, in which last situation it simulates the pain of lumbago. It varies greatly in intensity and duration, and is seldom continuously present during the whole course of the disease. It is aggravated or, if absent, elicited, by deep inspiration, by coughing, sneezing, laughing, percussion, external pressure, and sudden change of position. The pain of pleurisy is commonly severe—sometimes so severe as to cause extreme dyspnœa and great distress, together with signs of collapse. As a rule, but not invariably, it disappears upon the occurrence of effusion, as indicated by the physical signs.

Cough is a common symptom of acute pleurisy. It is frequently present from the beginning, disappearing when effusion has taken place, and recurring as the fluid undergoes resorption. It is short, dry, and painful. Sometimes it is wholly absent. Expectoration is likely to attend the cough in the stage of resorption, when some catarrhal bronchitis develops in the expanding lung. The sputum is usually slight in quantity, and consists of small masses of tenacious mucus, occasionally slightly streaked with blood. Respiration is increased in frequency, and is superior costal in type, and shallow. These changes are at first due to the pain excited by normal breathing; later, to the compression of the lung by the effused fluid. Under both conditions the disturbance of respiration may amount to positive dyspnœa.

When the effusion is rapidly poured out, especially in persons previously in good health, dyspnœa is often urgent, and may prevent the patient assuming the recumbent posture; when, however, it takes place slowly, and particularly in feeble or cachectic individuals, it may fill the pleural sac and compress the lung to a condition of carnification, without causing dyspnœa, save upon exertion. At the beginning of the attack the patient usually prefers to lie upon the sound side; as effusion takes place he lies indifferently upon either side; but as it increases he is most comfortable lying upon the affected side.

The pulse is constantly accelerated; in severe cases its volume and tension are reduced, and it is often irregular both in force and rhythm—changes induced by the pressure of the effusion upon the heart and great vessels.

Pressure-points of tenderness corresponding to those of intercostal neuralgia are occasionally found, and in children areas of cutaneous hyperæsthesia phenomena which point to the implication of the intercostal nerves in the inflammatory process.

In almost all cases of pleural effusion, rapid loss of strength, early anæmia, and if the disease be prolonged, emaciation are observed. In cases attended with large effusions cyanosis is likely to occur, and is sometimes present to a striking degree.

The urine is diminished and concentrated during the accumulation of the effusion; as this undergoes resorption the urine is often greatly increased in quantity, and clear, and of low specific gravity.

Physical Signs.—The lesions of pleurisy, both in the plastic stage and in the stage of liquid exudation, seriously interfere with the function of respiration and constitute coarse anatomical changes which fully respond to the methods of physical examination. The application of these methods is not only necessary to precision of diagnosis, but also yields results that are, clinically speaking, exact and definite. It is necessary to consider the signs obtained by the application of each of these methods—namely, Inspection, Palpation, Mensuration, Percussion, and Auscultation—in turn, as indicating the structural changes and visceral displacements produced by pleurisy (*a*) at its onset, while there is as yet little or no fluid exudate; (*b*) when the effusion is of moderate amount; (*c*) when attended with large effusions; (*d*) when resorption of the fluid takes place without deformity of the chest; and (*e*) when retraction and deformity of the chest follow the disappearance of the effusion. With the exception of a few minor points, which will be dwelt upon in the discussion of empyema, the physical signs are the same in any given case of pleural effusion whether the exudate be sero-fibrinous, purulent, or hæmorrhagic.

Inspection.—(*a*) At the onset of the attack, prior to the accumulation of fluid, the lesions are simply those of ordinary dry or fibrinous pleurisy, and the physical signs are those of that condition. Hence on inspection we note increased frequency of respiratory movement, in part due to the fever and in part to compensate for the shallowness of breathing in the desire to escape pain. This restriction of the respiratory excursion becomes manifest by comparison with the opposite side, and varies proportionately with the severity of the pain.

(*b*) When the effusion is of moderate amount, but not sufficient to occasion notable displacement of adjacent organs or changes in the contour of the thorax, inspection shows limitation of movement no longer due to pain (which may or may not be present), but to the mechanical impediment to the expansion of the compressed lung. If pain persist, the frequency of respiration continues, and often amounts to distressing dyspnœa. The effusion at this stage rises anteriorly almost to the level of the nipple.

(*c*) As the fluid increases, the respiratory movement on the affected side diminishes. It may be almost wholly absent. Dyspnœa is produced by excitement and movement of the body. The chest now bulges, especially in its lower and middle segments, and assumes in contour a modification of the inspiratory type. The lower intercostal spaces are widened. It is very rare that they bulge in in cases of purulent effusion. The antero-posterior diameter of the chest is increased; the nipple and scapula are more distant from the median line than upon the opposite side; the hypochondrium is prominent, the shoulder elevated, and the whole side appears larger than its fellow. In lean persons the displacement of the heart may be recognized upon inspection, the impulse in right-sided pleurisy being visible to the left of the nipple line in the fourth and fifth interspaces, or even in the line of the anterior axillary fold; and in cases of large effusion in the left pleural cavity, the impulse may be visible in the third and fourth intercostal spaces on the right side, in the nipple line or even beyond it.

(*d*) When the effusion is undergoing resorption, while as yet the chest is not retracted, and in those comparatively rare cases in which subsequent deformity does not occur, inspection shows a decrease in the volume of the affected side and in the prominence of the hypochondrium, with narrowing and retraction of the intercostal spaces, or a return of the respiratory excursion of the chest-wall, and a restoration of the visible beat of the heart to its normal position.

(*e*) The resorption of the effusion is, in a very large proportion of the cases, followed by retraction and deformity of the chest and displacement of adjacent organs toward the affected half of the thorax. This is a slow process, dependent upon the organization of the pleural exudate and the contraction of the new connective tissue thus formed, and of that owing its origin to associated inflammatory processes at the periphery of the lung. It requires several months for completion. While in children and adolescents the resulting deformity may be transient, in adults it is likely to be permanent. In some instances there is general retraction of the side; in others there is a circumscribed depression, and very often these two conditions coexist. The chest assumes an exaggerated modification of the exspiratory type. Its volume is diminished, its antero-posterior diameter greatly decreased; the lower intercostal spaces are narrowed until the ribs are in contact; the shoulder is lowered; the nipple sinks and approaches the median line, and the inner border of the scapula stands out from the surface of the chest. There is commonly lateral curvature of the spine, the concavity looking toward the affected side; but some cases remain free from this deformity, and in a small proportion of cases presenting advanced contraction the convexity looks toward the affected side. An extensive shallow depression at the base of the affected side of the chest anteriorly may exist without general contraction, and it is not rare that a history of old pleurisy is obtained in patients who present a central cup-like depression in the region of the ensiform cartilage.

Palpation.—(*a*) Prior to the effusion the sense of touch will in many cases appreciate a distinct friction—pleural fremitus—corresponding in location with the friction-sounds heard on auscultation, and in rhythm with the respiratory movements. Diminution of vocal fremitus is among the early signs of an accumulating effusion. (*b*) In moderate and (*c*) in large effusions, palpation confirms certain of the physical signs revealed by inspection. Among these are the restricted respiratory movements of the chest-wall, the enlargement of the thorax, the widening and obliteration of the intercostal spaces, and the abnormal position of the cardiac impulse. The last may very often be determined by palpation when it is not appreciable upon inspection. Fluctuation is scarcely ever present; it cannot be regarded as a sign of pleural effusion under ordinary circumstances. Œdema of the chest-wall is almost equally infrequent in sero-fibrinous pleurisies. In small effusions the vocal fremitus is enfeebled; if the amount of fluid be large, vocal fremitus is greatly diminished or absent altogether. The fact is not to be overlooked that normally, as a rule, the voice-vibrations are more distinctly felt upon the surface of the right than on the left side of the chest. This physical sign is directly related to the strength and pitch of the voice. It is hence of more value in men than in women, and least of all in children. It may be studied in infants during the act of crying, and it is worthy of note that it may be present in children over an effusion of considerable amount. The vocal fremitus may be conducted to the chest-wall by the way of localized pleural adhesions. It is also occasionally conducted for some distance across the chest (posteriorly) from the sound side by the medium of the chest-wall itself, and under these circumstance may become a source of momentary doubt as to the diagnosis.

(*d*) As the effusion undergoes resorption, one may recognize, by palpation even more accurately and satisfactorily than by inspection, the change in contour and movement of the chest-walls, the recession of the intercostal spaces, and the gradual return of the heart, liver, and spleen to their normal positions. The vocal fremitus may again be felt with increasing distinctness, and, until adhesions take place, the fremitus of pleural friction.

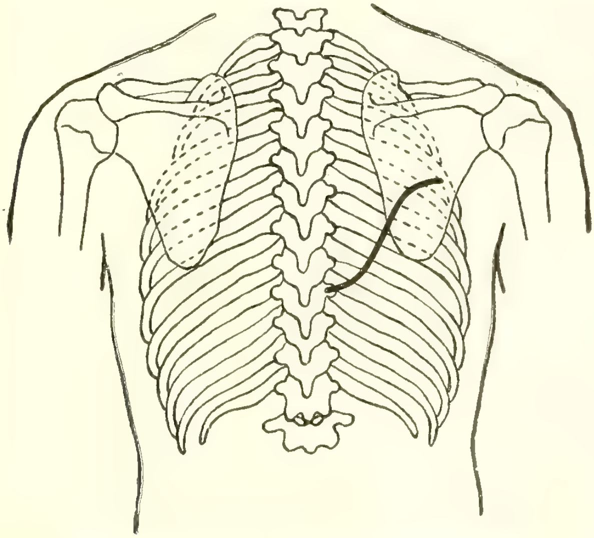
(*e*) Finally, when deformity follows resorption, palpation detects with precision the limited movement of the affected side, the changes in the contour of the chest, the spinal deviations, the approximated ribs, and the local recession of the wall of the thorax. Vocal fremitus is frequently somewhat intensified.

Mensuration.—(*a*) Before effusion has taken place, and (*b*) while it is moderate in amount, the tape shows the difference in inspiratory expansion between the two sides. In right-handed adults the right side is naturally slightly larger than the left. Allowance being made for this difference, actual measurement shows no increase in the size of the affected side, nor does the cyrtometer show alteration in its contour. (*c*) When, however, the effusion is large, the circumference of the affected side is found upon measurement to exceed that of the sound side by an inch or more—an increase which is less than seems apparent upon inspection, and which does not indicate the amount of the effusion, which finds place for itself by compressing the lung and displacing the diaphragm downward and the mediastinum toward the opposite side. A decided difference in the horizontal outline of the two sides are shown by cyrtometric tracings. (*d*) These conditions disappear when resorption of the fluid takes place without deformity; and (*e*) when general retraction takes place, they are replaced by shortening of the diameters, diminution of the circumference, and alteration of the outline of the affected side, which assumes in an exaggerated degree the peculiarities of the expiratory type. Actual measurement shows to a less marked extent similar changes in cases in which local deformities are present.

Percussion.—(*a*) Early in the attack, while the friction-sound is heard and the pain is severe, the results of this method of physical diagnosis are negative. At most, owing to shallow breathing, the percussion signs may indicate imperfect expansion of the margins of the lungs at the base. Nor are significant signs elicited until the effusion reaches half a litre (500 cc., one pint). The resonance is first impaired at the base posteriorly, more rarely in the infra-axillary region, and, as the effusion increases, is replaced by dulness, which becomes more and more marked. (*b*) When the level of the fluid in front reaches to the fourth rib, the percussion-signs are characteristic. There is dulness below the upper level of the fluid, toward the base rapidly progressing to absolute flatness.

If the finger be used as a pleximeter, the absence of vibration is not less notable than the want of resonance, which is much more complete than in pneumonia. Above the line of dulness the percussion-sound is tympanitic or vesiculo-tympanitic from loss of normal tension in the pulmonary tissue. This change of quality, known as Skoda's resonance, extends some distance above the line of dulness posteriorly and laterally, but in the ordinary semi-recumbent posture of patients in bed it rises in front to the apex, and constitutes in the infraclavicular region a very important sign. When the patient is in the erect posture the upper line of dulness is not horizontal, but is higher behind

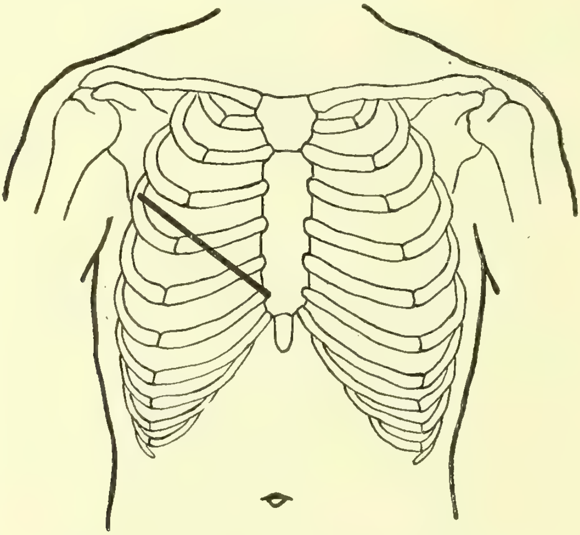
FIG. 39.



Upper line of Flatness posteriorly; moderate effusion.

and laterally than in front, forming an irregular parabolic curve. As first described by Ellis of Boston, "this curve begins with medium effusions relatively low down in the back, passes outward from the vertebral column, and soon turns upward, and proceeds obliquely across the back to the axillary region, where it reaches its highest point. Thence it advances in a straight line, but with a slight descent, to the sternum." Garland has pointed out the resemblance of this curved line to the italic letter *S*. He states that it "may be traced, by proper percussion, in any case of free, uncomplicated pleurisy when the patient's body is erect and the amount of fluid present is not excessive. As any effusion increases in amount, the curve of its distribution gradually rises and tends to flatten out, so that it no longer presents its characteristic *S* feature after it reaches the second rib. At this point, when the fluid occupies nearly the entire side, the curve comes quite near to the horizontal; but if some of the fluid be withdrawn by aspiration or absorption, the letter *S* curve will reappear, and retreat downward in the inverse order of its advance, until with entire absorption it becomes merged with the normal boundary of the lung." This curve is modified by pleural adhesions and by pathological conditions of the lung itself, among which may be named pneumonic consolidation, tuberculous infiltration, and pulmonary emphysema. In order to mark out Ellis's curve, precussion should be performed lightly and in lines at right angles to the line of flatness, and attention must be given to the distinction between the relative dulness in the triangular space between the curved line and the vertebræ and the absolute dulness (flatness) beyond and below that line. The line of dulness is not displaced by deep inspiration. In some cases it varies somewhat in accordance with changes in the position of the body, as from the sitting to the recumbent posture. This change of level takes place only

Fig. 40.



Upper line of Flatness Anteriorly; moderate effusion.

in effusions of moderate amount untrammelled by adhesions. It is much less common in the case of pleurisy than in that of the serous transudations of hydrothorax. To detect it a delay of some minutes after the change of posture is sometimes necessary, as the fluid slowly obeys the action of gravity. When present it is a positive sign of fluid in the pleural sac.

On the right side the dulness is continuous, without modification, with that of the liver; and on the left it extends in the mammillary line to Traube's semilunar space, which it invades.

(*c*) Very large effusions, distending the wall of the chest and leading to displacement of adjacent organs, give rise to flatness over the greater part of the affected side. Diminished resonance, the quality of which is tympanitic, may be present above the spine of the scapula, and an exquisite tympanitic percussion-sound may be elicited immediately below the clavicle. In the latter region it is not rare to discover the cracked-pot sound—*bruit de pot fêlé.* In the largest effusions the resonance disappears from these localities, and there remains only a limited area of obscure resonance over the compressed lung at the upper part of the chest near the spine. The level of the flatness then rises to the clavicle, and may extend across the upper portion of the sternum, reaching to or even beyond its opposite border.

In right-sided effusions the lower margin of liver-dulness is depressed, and may reach the level of the navel. The displacement of the left border of the heart may be made out by percussion. In left-sided effusions the semilunar space of Traube is encroached upon or even obliterated, so that its tympanitic percussion-sound is replaced by flatness; the spleen is displaced downward, and the cardiac dulness extends an inch or more to the right of the right border of the sternum. In large effusions the respiratory displacement of the organs in relation with the diaphragm—namely, the liver in right-sided pleurisy and the cardiac extremity of the stomach and the spleen in left-sided effusion—is absent. (*d*, *e*) When resorption takes place, the signs which the presence of the effusion and the displacement of organs yield upon percussion disappear in the inverse order of their appearance, leaving, upon retraction of the affected side, general impairment of resonance and other percussion-signs indicating the final encroachment of the mediastinum (originally displaced toward the opposite side) upon that side of the thorax which was the seat of the morbid process.

Auscultation.—(*a*) Early in the attack the vesicular murmur of the affected side is, owing to the shallow breathing, of diminished intensity and duration. The rhythm of the respiration is disturbed in consequence of the pain, and the breathing becomes irregular and jerky. In the course of a few hours there may be heard a fine friction-sound. At first circumscribed, this sign may be readily overlooked. It is to be sought in the lower zone of the chest, in the inframammary, the infra-axillary, and infrascapular regions. In one or the other of these areas the friction-sound is often diffuse, or it may be circumscribed at several points. It usually occurs at some period of both respiratory acts, but is commonly more distinct during, and especially toward the end of, inspiration than during expiration. It may be absent during the latter movement. Occasionally the rub is broken or interrupted during the inspiratory act, and not infrequently it ceases to be heard during one or more respirations. The character of the sound is most variable. It may be fine and grazing, or it may resemble the crepitant râle of pneumonia—a resemblance increased by the fact that it is frequently most distinct at the end of inspiration. Or, again, it is coarse, like the creaking of new leather, or it may be defined by such terms as grating, scraping, or sawing. Pleural friction occasionally resembles intrapulmonary râles other than crepitant, and it is frequently associated with various bronchial râles due to coëxisting bronchitis. The friction-sounds are more superficial—that is, they give the impression of being produced nearer to the ear—and they are less modified by cough and deep inspiration. When the border of the lung adjacent to the pericardium is involved, there may sometimes be heard a pleuro-pericardial friction-sound. (*b*) As the fluid accumulates the breath-sounds become feeble and distant; vesicular respiration gives place to indistinct broncho-vesicular breathing, which in turn is replaced by bronchial breathing, and this often, especially in children, has an amphoric or cavernous quality. In small effusions deep breathing may generate crepitant râles by the inflation of compressed vesicles at the periphery of the lung. Above the level of the effusion the respiratory sounds are broncho-vesicular (harsh), and the vesicular murmur of the opposite side is intensified (puerile).

The vocal resonance over the effusion is diminished or absent; there may be bronchophony, and at the upper border of the liquid the voice occasionally has the bleating quality called ægophony.

(*c*) In large effusions the signs upon auscultation vary according to the degree of compression to which the lung is subjected. When the effusion is very large and the lung is fully compressed, the breath-sounds wholly disappear both in front and behind, except an indistinct bronchial respiration over a limited area in the interscapular region along the spine, and in extreme cases even that may be absent. Vocal resonance is lost. Baccelli, however, has called attention to the fact that the whispered voice is sometimes distinctly transmitted through a serous effusion, but is not heard through pus. When the cardiac impulse cannot be felt, auscultation enables us to determine the position of the heart by the situation in which the sounds are heard with maximum intensity. A systolic murmur may sometimes be detected, which disappears upon the aspiration or resorption of the fluid.

(*d*, *e*) The resorption of the effusion is attended with the reappearance of the friction-sounds, louder and more diffuse than at first, and the return of the breath-sounds in the reverse order of modification in which they were suppressed. For a varying period, often extending over many months, the respiratory sounds at the base continue to be feeble and indistinct. As the compressed pulmonary tissue expands, coarse crepitus of a peculiar creaking character is frequently heard over the upper part of the lung. This is not associated with cough and expectoration, and after a time disappears. The phenomenon is doubtless due to the expansion of the previously compressed lung, and its mechanism is that of the crepitus which attends the expansion of atelectatic lung-tissue under other circumstances. It is not to be confounded with the crackling of early pulmonary tuberculosis, which develops in a group of cases in the course of pleurisy. As the effusion subsides, bronchophony reappears, and at points the voice has a peculiar nasal twang or there is well-marked ægophony. Auscultation enables us to follow the return of the heart-sounds to their normal position.

The **course** of acute sero-fibrinous pleurisy is not regularly defined. The fever may last a week or ten days, exceptionally longer. Early in this period the cough becomes less troublesome, the pain disappears, and effusion takes place. The effusion may form rapidly ; as a rule, however, it accumulates gradually. Many acute effusions of small amount undergo rapid resolution. In larger effusions, reaching as high as the fourth rib in front, resorption takes place slowly and may occupy a period of weeks. In large effusions, reaching to the clavicle, spontaneous resolution takes place rarely and very slowly ; nevertheless, effusions of considerable size, which have persisted for months, may undergo resolution without surgical intervention.

A sero-fibrinous effusion may persist indefinitely without undergoing great change, and, especially in tuberculous cases, may reappear after repeated aspiration. In other cases the effusion becomes purulent. The resorption of the fluid results in the formation of adhesions, which for a time give rise to pain upon exertion and upon deep breathing, but which ultimately, when recovery is otherwise complete, cease to occasion inconvenience. The retractions and deformities of the chest which occur in convalescence have already been described. Death is comparatively infrequent in acute primary sero-fibrinous pleurisy. An unlooked-for fatal issue may occur even in cases of moderately large effusion, in consequence of syncope following slight sudden exertion. This accident has been attributed to embolism or thrombosis of the heart or pulmonary artery, degeneration of the heart, or sudden œdema of the opposite lung. Bartols and Fraentzel ascribe it, in some of the cases, to sudden twisting of the inferior vena cava, caused by the dislocation of the heart. Acute sero-fibrinous pleurisy occurring in the course of advanced chronic disease, as nephritis or pulmonary tuberculosis, may rapidly lead to death.

(*b*) PURULENT PLEURISY.—**Synonym.**—Empyema.

The term "pyothorax" should be reserved for those comparatively infrequent cases in which pus enters the pleural sac from the rupture of an abscess in neighboring structures, as in the course of caries of the spine or abscess of the liver. Purulent pleurisy is at once excited and empyema results. The distinction is an artificial one.

Etiology.—The effusion of pleurisy is frequently purulent. This happens (*a*) when a sero-fibrinous effusion is infected by pyogenic micro-organisms through the chest-walls or from within the body, and (*b*) when the pleural inflammation is originally excited by pyogenic organisms.

A sero-fibrinous pleurisy may be converted into an empyema in consequence of a penetrating wound, fracture of a rib, and of the operation of aspiration or other surgical procedure conducted without due regard to cleanliness. In certain cases of tuberculous pleurisy, after repeated aspiration of sero-fibrinous fluid, nearly transparent or but slightly turbid, the accumulation finally becomes purulent.

Other tuberculous pleurisies, infections perhaps of greater intensity, are purulent from the outset. Those which follow the necrosis and rupture of the wall of a subpleural tuberculous cavity are invariably so. Purulent pleurisy results from the access of pus to the pleura from the rupture of a retrosternal abscess or an abscess of the liver or in caries of the ribs or vertebræ. Infection may extend to the pleura in peritonitis or in malignant disease of the lung or œsophagus, or in abscess and gangrene of the lung, unless the pleural surfaces have early formed extensive adhesions. This variety of pleural inflammation occurs frequently as a secondary affection in the acute infectious diseases, especially croupous pneumonia, diphtheria, and scarlet fever; less commonly in typhoid fever, measles, influenza, and whooping-cough. It occurs also in connection with puerperal infection.

No period of life is exempt from the liability to empyema. It is especially common in the very young. Nearly one-third of all cases of effusion into the pleura in childhood are purulent, and it has been estimated that suppurative pleurisy is more prevalent during the first five years than at any subsequent period.

Morbid Anatomy.—The effusion may be general or encysted. In children it is usually general. In old empyemata the lung is thoroughly compressed and carnified, all traces of alveolar structure being lost. It occupies the upper and posterior part of the thoracic cavity as a flat mass of airless tissue. The pleura is sometimes little thickened, especially in recent cases; but when the empyema has existed for some time the pleura becomes tough and leathery, and is greatly thickened. This condition of the pleura, which is present not only in that layer which lines the chest, but also in that which overlies and imprisons the compressed lung, serves as an effectual barrier to the expansion of the lung under any circumstances, and constitutes an urgent indication for the radical treatment of empyemata at an early stage of their development. After a time, as a result of pressure or in consequence of the localized intensity of the inflammation, local necrosis of the pleura may take place. This results in perforation of the sac and escape of the pus in various directions. Thus the fluid may pass into the lung by an extended surface, by soakage, or by the way of a fistula communicating with a bronchus; it may find its way out through an intercostal space, and form a tumor beneath the integument—*empyema necessitatis*. The perforation may take place at various parts of the wall of the chest; more commonly it is in front and between the third and sixth ribs and the border of the sternum and the line of the anterior axillary fold, the most frequent position being in the fifth interspace. The rupture of the tumor which constitutes empyema necessitatis may result in a direct fistulous communication with the pleural cavity, or the pus may burrow some distance beneath the skin and then escape, thus causing an elongated oblique sinus. The occurrence of either a pulmonary or a costal fistula is likely to be followed by pneumo-pyothorax. When the tumor is situated in the præcordial space it may exhibit pulsations which are synchronous with the heart's action. Empyema necessitatis is usually single; exceptionally two or more tumors are formed. This condition is more frequent in childhood and early life than at a later age. In rare cases the pus may perforate the diaphragm and reach the peritoneum, setting up peritonitis, or, if shut off from the cavity of the peritoneum by adhesions, it may find its way to the surface at distant and unlooked-for points. It may escape directly into the stomach or into the œsophagus, and cases have been recorded in which an empyema has ruptured into the pericardium. Finally, the pus may work its way down the spine and along the psoas muscle into the iliac fossa. Both the visceral and costal pleuræ show patches of erosion, and in old cases fistulous tracts communicating with a bronchus or through the chest-wall are often observed.

The exudate is variable. In some cases it is sero-pus, only moderately turbid and containing fibrin-masses; in others it separates after death into a supernatant greenish-yellow, nearly transparent, serous fluid, and a thick, yellowish or greenish purulent fluid which gravitates to the lowest portions of the cavity of the chest; or, again, it consists of a uniform, thick, creamy pus. Its odor is commonly simply mawkish and unpleasant; occasionally it is foul, especially in traumatic cases and neglected pneumothorax; and in gangrene of the lung it is always intensely offensive. The odor is likely to be horribly stinking in those empyemata which occur in puerperal and other forms of septicæmia, and in such cases the pus is of the kind called ichorous.

The facts brought to light in regard to empyema by bacteriological research are extremely interesting and important. The infection may be either I. simple, or II. mixed.

I. Simple infection of the pleural exudate.

A single agent of suppuration only is present in the exudate, which is so to speak, a pure culture.

A. Common forms:
- (*a*) Purulent pleurisy from infection by pneumococci;
- (*b*) From infection by streptococci;
- (*c*) From infection by tubercle-bacilli.

B. Rare forms:
- (*a*) Purulent pleurisy from infection by staphylococci;
- (*b*) From infection by Eberth's bacilli.

II. Mixed infection of the pleural exudate.

Two or more pyogenic microbes are found together in the exudate.

A. Common forms:
- (*a*) The exudate contains tubercle-bacilli and streptococci;
- (*b*) Streptococci and staphylococci;
- (*c*) Streptococci and saprophytic bacteria (microbes of gangrene).

Symptomatology.—The symptoms of purulent pleurisy are to some extent dependent upon the nature of the infection as indicated by its bacteriological characters. Debove and Courtois-Suffit have not only made the special infection a basis for the classification of the cases, but they have also erected upon it a systematic arrangement of the indications for treatment. At the risk of some repetition it is desirable to study the symptomatology from that point of view.

Acute forms:

Purulent Pleurisy from Infection by Pneumococci.—This form of empyema may be primary; much more frequently it is secondary. It then occurs in connection with pneumonia, or, less often, with peritonitis or meningitis. It is common in children and in adults between the ages of twenty and thirty years. Later in life it becomes progressively less frequent.

When this form of pleurisy is primary—and this is true of primary acute pleurisies in general—the onset is abrupt and attended with acute febrile movement, considerable pain, and the signs of rapid effusion.

When it is secondary to pneumonia it may develop during the course of the primary disease or during the convalescence. The access of fever is rapid, the temperature quickly reaching 104° F., and varying between 100.5° and 104° F. The removal of the fluid is followed by a rapid decline to the normal. If, however, the effusion re-forms, the temperature rises as before. Repeated chills and hectic phenomena are absent from this form of infection. If they occur, they must be ascribed to secondary infection, usually by streptococci pyogenes, or to the local necrosis of the pleuræ which precedes the formation of a bronchial fistula or an empyema necessitatis—events which are especially frequent in empyema due to the pneumococci. The volume of the effusion varies from two to five or even six litres. It cannot be regarded as a measure of the gravity of the case. In very rare cases the effusion, purulent though it be, undergoes resorption; very frequently it becomes encysted, and in a considerable proportion of the cases it has been spontaneously evacuated by way of a bronchial or thoracic fistula.

Purulent Pleurisy from Infection by Streptococci.—This group of cases includes the greater number of secondary empyemata. The pleural inflammation may arise during the course of the primary disease or after it. In the former case the empyema is insidious in onset, its symptoms being masked by those of the primary disease, and it may remain latent for a considerable time after being discovered in the course of a routine physical examination. If the pleurisy be primary, which is very rare, or if it occur after recovery from the primary disease, the onset is abrupt, and fever and pain are prominent symptoms. The rise of temperature is rapid and considerable. It may reach 105° F. or higher. After a few days it falls to 100° F. A rise quickly succeeds, and the subsequent range is characterized by the irregular and extreme exacerbations and remissions which attend the fever of suppuration. The withdrawal of the pus is followed by a fall of the temperature, which after a day or two rises again, coincidently with the recurrence of the signs of reaccumulation of the effusion.

The constitutional symptoms are severe. They consist of repeated chills, profuse sweating, and grave disturbances of digestion and nutrition. The quantity of the effusion is usually moderate, not often exceeding three litres. The tendency of the effusion to reaccumulate is characteristic of this form of empyema, which does not undergo resorption, and very rarely is spontaneously evacuated either by a bronchial fistula or by the formation of an empyema necessitatis.

Purulent pleurisy from infection by Eberth's bacillus (bacillus typhosus) alone is extremely rare. The empyema which occurs in the course of typhoid fever is, in the great majority of the cases, due to streptococcus-infection, and presents the symptoms already described as occurring in that form of the disease.

Purulent pleurisy due to simple infection by staphylococci is likewise extremely rare. Cases in which these organisms are encountered in association with other pyogenic bacteria are common. In the mixed infections the symptoms are those of the more severe elements of infection. Thus the comparatively benign pneumococcus-empyema is, by accidental secondary infection of the effusion in exploratory puncture or aspiration, at once converted into the more dangerous streptococcus-empyema.

Gangrenous pleurisy occurs as a secondary affection in the sequence of gangrene of the lung. It is occasionally encountered in connection with embolism and gangrenous infarction in the acute infectious diseases and in septic pneumonia.

Putrid pleurisy may arise independently of any special disease of the lung. Saprophytic bacteria may find access to an existing effusion in operative procedures, or may infect the pleura at the same time with the more common pyogenic bacteria, as in the case of abscess of the lung or other adjacent organ. They may likewise reach the pleura by the rupture of a pulmonary abscess or of a subpleural vomica. The organisms by which these forms of pleurisy are excited are various. The symptoms are grave. If the onset is sudden, there is intense and prolonged pleural pain. The fever rapidly reaches a high range. It is attended with moderate morning remissions, followed by intense exacerbations ushered in by chills and followed by colliquative sweating. Evidences of profound toxæmia quickly appear. There is extreme prostration; the face is pale, often cyanotic. The tongue quickly becomes dry and brown; vomiting is frequent and uncontrollable; diarrhœa is present.

Any of the foregoing forms of purulent pleurisy may occur in a tuberculous individual. They present no especial feature under such circumstances, save that their course is unfavorably influenced by the previous illness of the subject. They cannot be regarded strictly as tuberculous pleurisies. Those empyemata which are due to tuberculosis of the pleura are essentially different from the forms described, and merit separate consideration.

They begin as a local tuberculous process, either of the pleura or of the lung. In the latter case they come on early in the disease, very often without previous symptoms, sometimes after an attack of hæmoptysis. Tuberculous pleurisy runs a chronic course. Its onset is usually insidious, and its evolution is very often marked by the mildness of the symptoms. In other words, it may for a long time be latent. The effusion is at first sero-fibrinous. It not rarely undergoes resorption, so that the patient is able to resume his occupation. But he does not regain his health. Little by little, shortness of breath returns and oppression increases. While quiet the patient breathes well enough, but the slightest exertion brings on dyspnœa. Meanwhile nutrition fails and anæmia becomes pronounced. Physical examination now reveals a large effusion, which upon aspiration is found to be sero-purulent or purulent. Again the symptoms improve, and for a time, sometimes extending over months, the patient is able to follow his ordinary avocation. But he is not well, and the reaccumulation of the effusion demands repetition of the aspiration at various intervals. At length the pulmonary tuberculosis, to which pleural tuberculosis tends or with which it is associated, shows itself and runs a more or less rapid course to the fatal termination, or acute tuberculosis develops, or the patient succumbs to the accidents of pulmonary tuberculosis, such as pneumothorax or a terminal blood-spitting.

It is a remarkable fact that in a large proportion of the cases of chronic empyema which are tuberculous the exudate is sterile. Culture-experiments show neither the tubercle-bacillus nor the microbes of suppuration. Nevertheless, the development of tubercle has, in animals, frequently followed the intraperitoneal inoculation of this apparently sterile fluid.

Physical Signs.—The physical signs of empyema are practically those already described as present in sero-fibrinous effusions. A few special points require mention.

The paresis of the muscular wall of the chest is greater in empyema than in sero-fibrinous pleurisy. The diaphragm sinks lower, the intercostal spaces are more likely to bulge, and the mediastinum retreats to a greater degree before the effusion, so that the heart is dislocated to a greater extent. The chest is more decidedly enlarged in empyema. The integuments are more frequently œdematous and the superficial veins are enlarged. These signs are, as a rule, more marked in children and adolescents than later in life. In children distinct bronchial respiration is sometimes heard over a large purulent effusion. Whispering pectoriloquy is, as a rule, to which there are numerous exceptions, not heard over purulent effusions—the sign of Baccelli.

Pulsating pleurisy constitutes a curious variety of pleurisy with effusion. The phenomenon has long been known, though the number of recorded cases is limited. I have had the opportunity of studying three cases. In one only among the recorded observations was the effusion serous. In the others it was purulent. In all but three or four it occupied the left side. The characteristic phenomenon is pulsation synchronous with the cardiac impulse. The cases may be arranged in two groups: 1, intrapleural pulsating pleurisy, in which the pus is confined within the pleura and the pulsation is diffuse; and 2, pulsating empyema necessitatis, characterized by a subcutaneous pulsating tumor. The cases are usually of long standing. Pneumothorax may be present. The heart is usually much displaced, and the lung upon the affected side greatly compressed. Various theories have been advanced to explain this curious phenomenon, none of which is, however, sufficiently comprehensive to account for all of the cases.

Other varieties which require separate consideration are—

INTERLOBAR PLEURISY.—It is probable that this interesting and not very uncommon variety is a sequel of an antecedent general pleurisy, in the course of which adhesions have been formed which cut off the interlobar space from the general pleural sac. It is more frequent on the right than on the left side, and in children than in adults. The abscess thus formed is frequently situated between the upper and middle lobes, near the root of the lung. It varies in size from an egg to an orange, but may attain the size of the fœtal head. It commonly, after a period of ill-health without definite symptoms or of chest-symptoms not associated with definite results from ordinary methods of investigation, ruptures into a bronchus. If the abscess be of considerable size, it presents the signs of an abscess of the lung, for which it may readily be mistaken.

DIAPHRAGMATIC PLEURISY.—The pleurisy involves and may be limited to the diaphragmatic surface. It may be dry or there may be effusion either sero-fibrinous or purulent. When present the effusion is usually of small amount. There are pain low down in the side, and tenderness upon pressure at the point of insertion of the diaphragm at the tenth rib. The remaining symptoms are those of an acute pleurisy. If the effusion be purulent, there may be œdema of the lower intercostal spaces, with bulging.

ENCYSTED PLEURISY.—The effusion, in consequence of adhesion, is circumscribed, or divided into two or more pockets which may or may not communicate. In addition to the interlobar and diaphragmatic surfaces, the encapsulated effusion may occupy various positions. It may be next the mediastinum near the root of the lung, or at the apex, or, again, it may be in the posterior or postero-lateral part of the chest. These conditions are very difficult to diagnosticate, and are frequently discovered not by clinical, but by anatomical diagnosis. The exploratory puncture should be employed in every suspected case.

(*c*) HÆMORRHAGIC PLEURISY.—Red blood-corpuscles may be detected in the exudate of any acute sero-fibrinous pleurisy. The term hæmorrhagic pleurisy is used to designate the cases in which there is an admixture of blood sufficient to be recognized by the naked eye. The term "hæmothorax" is applied to those conditions in which hæmorrhage into the pleura follows an injury to a larger intrathoracic blood-vessel, the pressure of a tumor upon the veins, or the rupture of an aneurism.

Etiology.—A hæmorrhagic effusion may occur in tuberculous pleurisy in consequence of the great vascularity of the tuberculous new-formation upon the pleural surface. It occurs also in malignant diseases involving the pleura, in cirrhosis of the liver, in the pleurisy of chronic nephritis, and in malignant cases of the acute infectious diseases. In rare instances the effusion is bloody in individuals previously in good health, and in whom, after aspiration, permanent recovery takes place. Blood may become admixed with the effusion in consequence of a wound of the pulmonary pleura inflicted by the needle of the aspirator, and it is likely to appear toward the end of the aspiration if the operation be unduly prolonged or the evacuation be too forcible.

Course and Terminations.—Plastic pleurisy runs, as a rule, a favorable course, and terminates anatomically in the formation of pleural adhesions; clinically, in recovery. Evidences of the presence of plastic or fibrinous exudation may be detected in the early stage of pleurisy with effusion; but the early presence of the signs of fluid determines the nature of the process. Sero-fibrinous pleurisy, which in a majority of the cases is unquestionably tuberculous, frequently terminates in spontaneous resorption and permanent recovery. In other instances recovery takes place after the aspiration of a portion of a large exudation, the relief of tension apparently being immediately followed by restoration of the function of the lymph-vessels; in other cases the withdrawal of a large part of the fluid, or even repeated aspiration, may be followed by expansion of the lung. In yet other instances the fluid becomes purulent, without immediate evidences of hectic, and the chronic empyema—which from its peculiar symptoms and course receives the designation of cold abscess of the pleura—arises. Under proper treatment recovery may occur under these circumstances. Nevertheless, the patients are in danger of developing pulmonary tuberculosis, and a large proportion sooner or later succumb to that disease.

Empyema is a very serious affection, the course of which depends greatly upon the nature of the pyogenic micro-organisms to which it is due. Cases due to simple infection by the pneumococcus must be regarded as benign; those caused by saprophytic bacteria as in the highest degree unfavorable; while those arising from streptococcous infection occupy an intermediate position as regards their danger to life. Empyemata due to mixed infection manifest a gravity and persistence derived from the more virulent of the bacteria present in the exudate.

Small empyemata, especially those which are circumscribed, may undergo gradual resorption, with thickening of the pleura, deposition of lime salts, and local chest-retraction, the case ending in recovery. Recovery may also follow the evacuation of the pus by a bronchial or thoracic fistula. When the lung is perforated death may ensue from the flooding of the bronchial tubes. A fatal issue almost always follows rupture into the peritoneum.

Diagnosis.—Acute dry or plastic pleurisy, both in its primary and secondary forms, is recognized without difficulty. The stitch in the side, increased upon deep inspiration, and the friction-sounds suffice for the diagnosis.

The diagnosis of pleurisy with effusion involves two questions: First, as to the presence of an effusion; second, as to its nature.

The determination of the first of these questions is not attended with difficulty in massive effusions nearly or quite filling the pleural sac and causing displacement of adjacent organs. It depends upon a careful consideration of the physical signs as fully set forth in the foregoing pages under the caption of Symptomatology. In certain cases of moderate effusion not attended by displacement of organs, in which bronchophony and bronchial breathing are preserved over the area of dulness, the differential diagnosis between effusion and pneumonia demands circumspection. It is not in all cases so simple a matter as the textbooks indicate. As a general rule—which is, however, not without exceptions—pneumonia begins more abruptly than pleurisy, with an intense and prolonged chill, followed at once by high fever, dyspnœa, and, after a time, by rusty sputum. There are differences in the physical signs which are constant and characteristic. Flatness, which is the rule over an effusion, is in contrast with the dulness of pneumonia; there is in pleurisy a distribution of the percussion-signs different from that of those of pneumonia, the area of impairment of resonance in the former being bounded by an irregular parabola, beginning low down near the spine and abruptly rising, to reach its highest point toward the axilla, while the dulness in pneumonia not infrequently conforms to the outlines of a lobe, or is, at all events, limited by an interlobar sulcus. A sign of great importance in effusion is the diminution or absence of vocal fremitus. Yet the fact remains that the occlusion of a bronchus by a plug of tough, fibrinous exudate will in cases of pneumonia annul for the time being the fremitus over the corresponding area. In cases of doubt an exploratory puncture should be made. With proper disinfection of the instrument, the skin of the patient, and the hands of the operator—*i. e.* with *surgical cleanliness*—this procedure is not attended with danger. I am in the habit of using for this purpose the aspirator instead of a hypodermic syringe. The former is a much more efficient instrument for exploration, and if fluid be found it may at once be withdrawn, in part or wholly, without the parade and circumstance of a second operation for that purpose.

Hydrothorax when unilateral, as is not infrequent in cardiac disease, presents the physical signs of an effusion due to pleural inflammation. The absence of friction-sounds, of stitch in the side, and of the rise in temperature which occur in acute pleurisy are of diagnostic importance. If the effusion be large, aspiration may be performed, and the question whether the fluid be an exudate or a transudate be determined by its chemical characters. On the right side abscess of the liver and hydatid cyst; on the left a large pericardial effusion; on either side an intrathoracic tumor, may simulate pleural effusion. The differential diagnosis in such cases must be made by a systematic and painstaking analysis of the physical signs, taken together and studied in relation to the symptoms and history of the case. These and other conditions not only present many of the signs of pleurisy with effusion, but they also very frequently cause that affection, so that we encounter the association of pneumonia, pericarditis, various tumors of the thorax and of the right lobe of the liver, or malignant disease of the lung and pleura, with fluid exudate in the pleura—conditions which are often not less serious than difficult of diagnosis.

The question as to the presence of fluid being settled in the affirmative, that of its nature may be decided by the use of the aspirator-needle in exploratory puncture. Not only should this be done in every case of doubt, but it should also be done without loss of time in every large effusion and in all cases in which resorption is unduly delayed or in which the constitutional disturbance or the chest-symptoms, especially those relating to the circulation, become urgent. The importance of bacteriological investigation of the fluid is not to be overlooked.

The special micro-organism or micro-organisms present can be determined by staining and culture; their virulence by inoculation-experiments upon animals.

Clinically, persistent fever, especially with great oscillations of temperature, sweating, and rapid anæmia, will arouse a suspicion that the exudate is purulent. This suspicion is, however, not always confirmed. In fact, the reverse is occasionally found to be the case, especially in children—namely, that pus is present with little or no fever, and in absence of sweating, and with only moderate impairment of the general health. Œdema of the chest-wall is commonly indicative of the presence of pus in the pleural cavity.

Empyema necessitatis is to be distinguished from subpleural abscess by the presence of the physical signs of an intrathoracic effusion, by the decrease of volume of the tumor by pressure, and by the fact that it diminishes upon inspiration and increases upon expiration. Subpleural abscess may occur in association with empyema, especially in children. Its resemblance to empyema necessitatis, great as it is, is merely superficial and the diagnosis is not difficult. Such an abscess may, by the establishment of a fistulous communication with the pleural cavity, be converted into an empyema necessitatis.

Pulsating empyema has been mistaken for aneurism of the aorta. Only in exceptional cases does the differential diagnosis between these two conditions present serious difficulty. Aneurism of the aorta bears a definite relation to the central long axis of the chest; its invasion of regions of normally clear percussion is circumscribed; it is usually the seat of murmurs or other sounds synchronous with the rhythm of the heart. The signs of pulsating empyema, whether intrathoracic or an empyema necessitatis, are, on the other hand, almost always upon the left side and at a distance from the median line; the dulness is uniformly at the base of the chest and extended, and murmurs are not present. Empyema necessitatis is influenced by pressure and respiratory movement of the chest-wall; aneurism is not.

Prognosis.—The prognosis of primary acute fibrinous pleurisy is favorable. That of the secondary form is dependent upon the nature of the primary affection. Chronic dry pleurisy will be fully described at a following page.

The prognosis of pleurisy with effusion depends largely, when the affection is primary, upon the character of the bacteria causing the process, and upon the promptness and thoroughness of the treatment. In sero-fibrinous pleurisy the prognosis as regards immediate recovery is favorable. A large proportion of the cases end in restoration to health, and many of them in permanent recovery. In empyema the outlook is much more grave. In simple pneumococcus-infection the mortality does not exceed 2 per cent.; in streptococcus and mixed infection it reaches 25 per cent. Tuberculous empyema is essentially a chronic affection, and the prognosis is more favorable as regards prolongation of life than restoration to health.

Secondary empyemata are of various significance. They add to the dangers of the processes in association with which they arise, and their occurrence is frequently, especially in septic diseases, the immediate herald of dissolution. The deformities and disabilities which follow cured empyemata are not always incompatible with the enjoyment of a fair degree of health and prolonged life.

Treatment.—DRY PLEURISY.—The treatment is symptomatic. The constitutional reaction is, as a rule, neither severe nor prolonged. The main indications are the stitch in the side and the restricted respiratory movement, which is largely due to the pain. The fever and the attendant derangement of the secretions may demand medication. The analgesic antipyretics, antipyrin, phenacetin, and acetanilid, fulfil here, as in other acute febriles states attended with pain, a double indication. Diaphoretics, as Dover's powder or mixtures containing solution of ammonium acetate, sweet spirit of nitre, and, if necessary, tincture of aconite, which are also diuretic, appear in many cases to be followed by favorable effects, and may sometimes be the means of averting effusion into the pleura. In all cases prompt purgation by means of a saline, or preferably by calomel, is called for. The pain in the side and the associated dyspnœa may be controlled by the hypodermic injection of morphine, which may be repeated from time to time in diminishing doses as the pain recurs. No other remedy for this purpose is at once so convenient and efficient. Morphine may be administered by the mouth or small doses of Dover's powder may be given at intervals. Leeching over the seat of pain is frequently followed by relief, and in suitable cases in children is especially useful. Six or eight leeches may be applied, and the abstraction of blood controlled as soon as the pain ceases. In young children not more than two or three leeches should be used. Dry or wet cupping is less used than formerly. These measures may be followed by mustard plasters, turpentine stupes, the application of stimulating liniments, or the temporary application of hot poultices. The last should not be continuously employed. The application of cold by means of ice-bags sometimes relieves pain, and may exercise a favorable influence upon the circulation of the inflamed pleura. The continuous use of cold compresses is inconvenient and unnecessary. Later, the pain may render it desirable to fix the side by means of overlapping strips of adhesive plaster applied as in the treatment of fracture of the ribs. Continuing pain often yields to flying blisters. If the symptoms are severe, the patient should stay in bed.

SERO-FIBRINOUS PLEURISY.—*Pleurisy with Effusion.*—The signs of the accumulation of a fluid exudate do not enable us to know its nature, nor do the constitutional symptoms in all cases supply definite criteria by which to determine this question. If the onset of the illness be moderately acute, the fever not intense and of short duration, sweating absent, and anæmia not marked, the presumption is in favor of a sero-fibrinous exudate, especially if the whispered voice be transmitted in auscultation. All these conditions may, however, obtain in the presence of a purulent effusion. For a time medicinal means may be employed with the hope of promoting the resorption of the fluid—a result which is fortunately common enough. These measures consist—

First, in the employment of systematic counter-irritation upon the affected chest. This may be accomplished by means of tincture of iodine, painted over a considerable surface, or, better, over a small area, night and morning until the skin becomes slightly inflamed, when a new spot is similarly treated; or a series of small blisters may be applied in succession.

Second, in the administration of drugs supposed to act directly in promoting resorption. Mercurials were at one time much used, but are now generally abandoned. Potassium iodide, until recently strongly advocated in the treatment of pleural effusion, appears to be falling into disfavor.

Third, in the method of elimination. Drugs acting vigorously upon the skin, the kidneys, and the intestinal tract are selected. The theory is that which underlies the use of these remedies in dropsy and anasarca. If the blood-serum is concentrated by depletion, the tendency to regain its normal consistency will lead to the absorption of the fluid from the tissues so long as fluid is given sparingly by the mouth. This mode of treatment has a basis of reason, and is often followed by prompt improvement. It may, however, readily be carried to an extreme, and therefore requires the exercise of caution.

Diaphoresis may be excited by the hot-air or vapor bath repeated daily, by the administration of salicylic acid or the salicylates, by antipyrin, and finally by the hypodermic injection of pilocarpine once a day. Due regard must be had to the depressing effect of all these measures upon the circulation and to the danger of the toxic effects of these powerful drugs.

Diuresis may be favored by the use of potassium citrate or acetate, and in children by sweet spirit of nitre. Digitalis is especially useful as a diuretic, and squill is employed with advantage. Infusions, mineral waters, and other medicaments which contain much fluid capable of acting freely upon the kidneys are contraindicated, as by supplying fluid to the blood they defeat the object for which the diuresis is provoked. The amount of fluid ingested must be carefully estimated and compared with the daily excretion of urine, which it should never be allowed to exceed, save when there is excessive sweating or profuse watery discharges from the bowels.

It was formerly the custom to use such active purgatives as elaterium, croton oil, large doses of colocynth or senna and the compound powder of jalap. These urgent and often injurious drugs have given place to salines in concentrated solution, which deplete the blood of its watery constituents with equal efficiency, more certainty, and less danger. For the systematic use of salines in dropsies and serous effusions the profession is indebted to Matthew Hay. Every morning or every second morning, according to the effect and the patient's power of endurance, from half an ounce to an ounce of magnesium sulphate is given in as little water as possible an hour before breakfast. Copious watery stools are produced. Rochelle salt may be used in the same manner.

To render this method of treatment effective it is necessary to reduce the quantity of liquid consumed by the patient to a minimum. Not more than ten ounces of fluid are allowed during twenty-four hours. It may consist of water or milk or a little wine or spirits well diluted. The diet should be composed of roast or boiled meats, dry bread, and eggs. Fruits and fresh vegetables are prohibited. Sugar, salt, except sparingly, and salted foods are to be avoided as unnecessarily increasing the privation arising from so great a restriction of fluid.

This *dry diet*, as it is called, should not be long enforced, especially in connection with the free elimination of fluid. If resorption begins in a stationary exudate, it usually proceeds rapidly. The diet, apart from the reduction of fluid, should be highly digestible and nutritious. In children a *dry diet* is scarcely practicable. Milk, meat-juices, the peptonoids, gruels, and wine or brandy are, on the other hand, to be generously supplied. Syrup of iron iodide is especially useful in the treatment of the pleural effusions of childhood. In adults iron is also useful during and subsequent to the stage of resorption, at which period digitalis, strychnine, and the vegetable bitters are indicated. Change of climate, an open-air life, gentle mountain-climbing, and respiratory gymnastics are desirable, especially for those predisposed to pulmonary tuberculosis.

If the effusion remains stationary or continues to increase, whenever it reaches to the level of the second rib, and promptly upon the supervention of such evidences of grave disturbances of the respiration and circulation as orthopnœa and cyanosis, an exploratory puncture must be practised. For this purpose the aspirator, as pointed out upon a preceding page, ought to be employed in preference to the hypodermic syringe. At a single operation we may then determine the presence of fluid, its character, and remove some part, or even the greater part, of it.

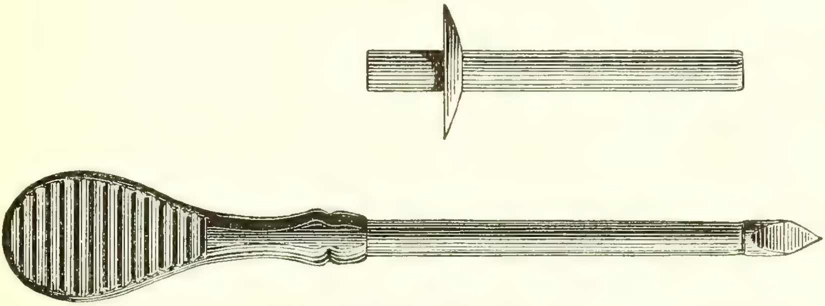
Immediate aspiration becomes imperative when the effusion is found to be complicated with pericarditis, valvular heart-disease, or pneumonia. Nor does fever constitute a contraindication. When the course of the temperature, the gravity of the symptoms, and the development of œdema of the chest-wall render the diagnosis of purulent pleurisy extremely probable, aspiration should be at once performed, with the purpose of securing the patient temporary relief and obtaining information concerning the character of the effusion, upon which to base future treatment. The dangers of aspiration properly performed are insignificant; those of delay in large effusions are of the gravest kind.

The operation is a simple one. Complete surgical antisepsis is obligatory. The point of election for the introduction of the needle is the seventh interspace in the line of the posterior axillary fold. The patient should assume the sitting posture, with the hand of the affected side upon the opposite shoulder, and should be supported by an assistant. The aspirator should in every instance be tested immediately before the operation. The needle—and the medium rather than the smaller sizes are preferable, even in exploratory operations—is to be guarded by the fingers against too deeply entering the chest, and thrust with a quick motion through the tissues just above the upper border of the rib. At this point there is no possibility of wounding the intercostal artery—a danger which, owing to its lying in a groove at the lower border of the ribs, is under any circumstances rather theoretical than practical. The possibility of puncturing the diaphragm and engaging the point of the needle in the liver on the right side and in the spleen on the left is to be considered, and avoided by advancing the needle slightly in an upward direction. Efforts to diminish the sensibility of the skin by the application of ice, sprays of ether, etc., the injection of cocaine, are attended with at least as much suffering as the puncture without such attempts at local anæsthesia, and unnecessarily prolong the operation. I therefore avoid them. The fluid should be slowly withdrawn, its flow being from time to time interrupted by compressing the conducting tube.

In large effusions, especially if recent, a litre or a litre and a half may often be slowly withdrawn without symptoms of distress. Beyond this amount caution is to be observed. Before and, if necessary, during the aspiration small quantities of wine or spirits may be administered, and at its conclusion a hypodermic injection of morphine. It is not good practice to push attempts at the withdrawal of the fluid to an extreme. In many cases the removal of a portion appears to relieve the lymphatic vessels of mechanical pressure and permit them to resume their functions. Among the indications for the immediate arrest of the process of aspiration are urgent dyspnœa, repeated and distressing cough, great pain, faintness or syncope, and the appearance of blood in the fluid. Sudden death has occurred during aspiration, an accident little likely to occur if the withdrawal of fluid is at once stopped upon the appearance of the above symptoms, and rare at any rate—far more so than in neglected large effusions. Cases have been described in which after aspiration great dyspnœa, attended with a profuse, thin, frothy sputum—*expectoration albumineuse*—has proved rapidly fatal. This accident has been attributed to œdema of the lung. It is extremely rare. The needle is to be withdrawn by a quick movement, as it was introduced. The finger is to be pressed over the point of puncture until a bit of adhesive plaster is applied. Recovery frequently follows a single aspiration. In other cases the operation must be repeated. If several repetitions are necessary at intervals, recovery is not likely to occur, even when for a long time the exudate remains only slightly turbid. After a while such cases—usually tuberculous—merge into empyema.

Purulent Pleurisy.—The treatment of empyema consists in free drainage and antisepsis. Cases due to simple infection by the pneumococcus, as shown by bacteriological examination of the pus, especially in infants, occasionally end in recovery in a short time after a single aspiration. Such cases not infrequently also get well after the discharge of the pus by way of a bronchial fistula. Notwithstanding these facts, we may lay down the rule that if the fluid be found upon exploratory puncture to be purulent and if after evacuation it reaccumulates, the empyema is to be treated as an abscess, without further delay, by free opening and drainage. For several years I have used for this purpose a short, wide trocar and canula. Upon the withdrawal of the former a soft catheter, No. 18, French, is introduced, and over this the canula is withdrawn. The catheter is retained in place by threads attached to the small safety-pin by which it is prevented from slipping into the cavity of the chest. These threads are attached to strips of adhesive plaster, and the side is dressed with a large mass of suitable absorbent antiseptic material, which is to be renewed once or twice a day. The catheter must be so arranged as to secure free drainage. The success of the treatment depends upon the thoroughness of the drainage. If, as the chest sinks in, the ribs nip the catheter, a portion of one or two of them must be excised. Irrigation is not necessary under ordinary circumstances; it is dangerous. I have seen a man seized with convulsions and die in a few minutes during cautious irrigation of the left chest, which had been practised from day to day for more than a week. Only in putrid or gangrenous empyema is irrigation justifiable. After a time the tube is shortened, and at length withdrawn. Retraction of the chest usually follows. Very often the cure is complete. The same result may be achieved by the free incision of an intercostal space. Pneumothorax of course at once occurs—a circumstance which need give no concern, as it is essential to the treatment and cannot be prevented. The cavity of the pleura, which is simply a huge abscess-cavity, closes by the gradual approximation of its walls; the lung expands, the mediastinum is drawn to the affected side, the diaphragm rises, the chest-wall sinks in. As the fistula caused by the operation, whether by the puncture of a trocar or by incision, must persist until the cavity is obliterated, it should be made at a higher level than is reached by the diaphragm in that process—

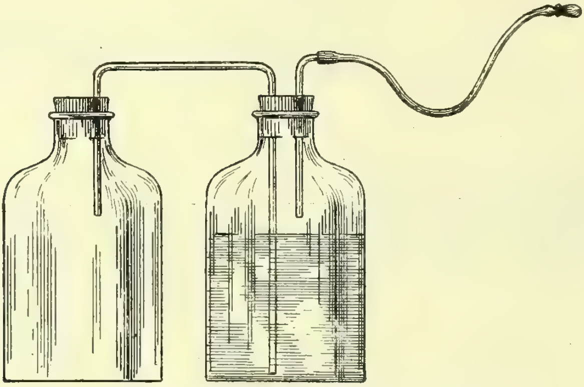
Fig. 41.



Trocar and Canula—actual size.

namely, in the fifth or sixth interspace. The drainage of the cavity and the expansion of the compressed lung are aided by systematic, frequent deep inspiratory efforts. The patient may do much for himself by the method of Ralston James, which has for some time been successfully used in the wards of the German Hospital. Two large bottles are fitted with rubber stoppers perforated for glass tubes. By blowing into one of them by means of a suitable mouthpiece, the water which it contains is forced into the other bottle. This process is repeated several times daily. The water should contain some disinfectant, and must be frequently changed. If the lung fail to expand, as is likely to be the case in old empyemata, the operation of Estlander should be performed by the surgeon. This operation consists in the resection of several ribs between the third and the eighth in the lateral region of the chest to the extent of an inch or more. It facilitates the approach of the chest-wall to the imperfectly expanded lung, and, while adding to the deformity, it diminishes or, in successful cases does away with, the pus-forming surface, and prevents or retards amyloid disease and the cachexia of suppuration. Estlander's operation yields better results in early than in later life.

Fig. 42.



Arrangement of Bottles for forced Expiration.

The presence of pus in the pleural sac constitutes an indication for operative interference, against which the gravest symptoms cannot be regarded as of weight. Free opening of the pleural cavity by the trocar or by incision has resulted in recovery in cases in which profound anæmia, extreme emaciation, hectic, dyspnœa, and a feeble and frequent pulse have seemed the indications of impending death.

II. Chronic Pleurisy.

As in the case of acute pleurisy, two anatomical forms are met with: 1, chronic dry pleurisy; and, 2, chronic pleurisy with effusion. The latter includes (*a*) chronic sero-fibrinous pleurisy, which is in the greater number of cases tuberculous; and (*b*) chronic empyema, which is sometimes due to streptococcus-infection, sometimes to mixed infection, to which the streptococcus imparts the chief peculiarities, and yet more frequently is tuberculous. Chronic pleurisy with effusion has been fully described in the foregoing pages.

Chronic dry pleurisy occurs as a sequel of pleurisy with effusion and as an independent affection—primitive dry pleurisy. Occurring after pleural effusions, it explains the retraction and deformities of the chest so common after recovery. As the pleural exudate is absorbed or upon its withdrawal, the layers of the pleura become agglutinated by the fibrin upon their surfaces, organization takes place, and a thick layer of connective tissue, with prolongations into the interlobular septa of the lung, is formed.

This altered and thickened pleura, the layers of which are completely fused, sometimes contains small cysts filled with clear fluid, and occasionally collections of inspissated pus in which lime salts have been deposited. The lung is not fully expanded, and in advanced cases remains compressed, permanently atelectatic, and is gradually converted into a fibroid mass. In young subjects the lung may after a time expand, the retraction of the chest disappear, and complete recovery follow. The process involves the base of the chest, and causes diminished respiratory murmur. It occurs after serofibrinous effusions and after empyemata. The greatest thickening of the pleura and the most marked deformity of the chest are encountered in cases of long-neglected empyema in which recovery has ultimately taken place. This condition is not incompatible with fairly good general health.

PRIMITIVE DRY PLEURISY.—Three forms are encountered:

First. Simple adhesions of the pleura, undergoing organization, uniting the pleural surfaces either in patches or extensively by layers of varying firmness or density, but not involving the connective-tissue framework of the lung or causing changes in the pulmonary tissue on the one hand, or deformity of the chest on the other. This form of primitive dry pleurisy may follow ordinary acute plastic pleurisy or may begin insidiously, being chronic from the outset. It may be primary or secondary. The pleurisy of pneumonia, which always gives rise to adhesions, is an example of secondary dry pleurisy of this form. When recent and extensive, this form of pleurisy causes a sensation of hindrance in deep breathing and uneasy dragging pains. Later it occasions neither morbid physical signs nor symptoms. Respiration is little interfered with in many cases in which upon post-mortem examination the adhesions are found to be universal.

Second. Tuberculous primitive dry pleurisy. This form is a constant accompaniment of chronic pulmonary tuberculosis. It progresses step by step as the process of tuberculization invades the lung. Hence it is common and usually most advanced at the apices, where the thickening of the fused pleural membranes may reach five or six millimetres. It is frequently universal. The interlobular septa are affected, and, together with tuberculous infiltration and caseation, there is always more or less fibroid change in the lung. One pleura only may be affected, or the process may be present in both. There is retraction of the chest-wall, more marked at those points which correspond to the greatest thickening of the pleuræ.

Another form of chronic tuberculous dry pleurisy is that in which the pleural surfaces are the seat of primary infection, the lung not being involved until late in the course of the disease. The pleura is more or less extensively studded with tubercles, and shows vascular, new-formed fibroid tissue; older, firm fibroid adhesions; and at points masses of caseating infiltrated tissue. One or both sides may be involved. This form of pleurisy is very rare.

Third. There is a form of primitive dry pleurisy with great thickening which requires separate description. It begins insidiously in persons of middle age. Most of the cases reported have occurred in males. There is persistent pain at the base of the chest, together with the signs of dry pleurisy. The affection is unilateral. Effusion does not occur. The area over which friction-sounds are heard becomes gradually more and more extended; the resonance at the base becomes impaired; there is feeble and distant breathing; respiratory movement is restricted; and progressive retraction of the side takes place. The general health is at first not greatly impaired. There is no elevation of temperature, and the pulse-rate is not accelerated. Nutrition, color, and energy may for years remain little affected. Intercurrent attacks of bronchitis occur, and chronic bronchitis attends the later course of the disease. Dyspnœa, especially upon exertion, is a prominent symptom. The urine is almost always slightly albuminous. The duration is from five to thirty years. The condition persists despite treatment. The pleura is greatly thickened, especially at the base. Its layers are fused, and it is continuous with dense and thick fibrous bands which traverse the sclerosed tissue of the lung. Bronchiectasis of all three varieties, globular, fusiform, and cylindrical, is encountered, though in some cases it has been absent.

Sir Andrew Clark[1] who has ably defended the opinion that this form of primitive dry pleurisy is non-tuberculous, arranges the pathological changes in four groups: Neo-plastic pleuritic membranes, the fibroid lung, the bronchiectatic lung, and the fibroid phthisical lung. These cases do not differ either clinically or in their pathological anatomy from the condition which on the one hand follows certain forms of pleural effusion, both sero-fibrinous and purulent, ending in recovery; or on the other hand, from that found in some of the cases of so-called pleurogenous chronic interstitial pneumonia. They cause alarm because they are often regarded as of tuberculous origin. In point of fact, the etiology of this group of pleurisies remains a matter of dispute. In some of the cases the presence of tuberculous infiltration of portions of the lung, with caseous softening and cavities, points to a process which may have been tuberculous from the beginning; in others neither tubercles nor tubercle-bacilli have been discovered. Nevertheless, there are those who regard this primitive chronic progressive fibrosis of the pleura and lung as, in a majority of the cases, a manifestation of tuberculosis.

PNEUMOTHORAX.

Synonyms.—Hydro-pneumothorax; Pyo-pneumothorax.

Definition.—Air in the pleural sac. Air alone is present in very rare instances. In the vast majority of cases the conditions are such that, together with the air, there is either a serous effusion (hydro-pneumothorax), or pus (pyo-pneumothorax). When blood is associated with air in the pleura, the condition is designated hæmo-pneumothorax.

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[1] *Lumleian Lectures*, 1885.

Etiology.—Air may find access to the pleural cavity (1) through a perforating lesion of the parietal pleura; (2) through such a lesion of the visceral pleura.

(1) Pneumothorax occurs (*a*) in penetrating wounds of the chest-wall; (*b*) in subpleural abscesses opening into the pleura and externally; and (*c*) in perforation of the pleura through the diaphragm, which occurs most commonly as a result of perforating ulcer of the stomach, with secondary abscess-formation, less commonly from malignant disease of the stomach or colon, and comparatively rarely in consequence of abscess of the liver. Perforation of the pleura may occur also in carcinoma of the œsophagus.

(2) The lesions of the visceral pleura which result in pneumothorax are—(*a*) Rupture of the lung from traumatism, especially contusions of the chest. (*b*) Spontaneous rupture of air-vesicles from straining; the air in the pleura may undergo resorption, and recovery take place; more commonly pleurisy and effusion result. (*c*) Rupture of emphysematous air-vesicles, either in acute emphysema, such as occurs in pertussis, or in the chronic form of the disease. (*d*) Gangrene of the lung, pulmonary abscess, hæmorrhagic infarct, hydatids, and cancer. These are among the rarer morbid conditions which cause pneumothorax. (*e*) Pulmonary tuberculosis; this is by far the most common cause—90 per cent. of all cases; the precise anatomical condition is local necrosis of the pleura involved in a caseating mass, or the rupture of the wall of a cavity situated at the periphery of the lung. (*f*) Perforation of the lung from the side of the pleura; this accident occurs only in empyema, and results in a bronchial fistula.

Whether or not pneumothorax may arise in consequence of the evolution of gas from a decomposing pleural exudate has been a matter of dispute.

Pneumothorax is more frequent in males than in females. It may occur at any period of life from infancy onward, but is much more common in adults.

Morbid Anatomy.—The condition is encountered upon the left side in a greater number of instances than upon the right. Not infrequently, owing to the valve-like action of the tissues at the seat of perforation, the air has accumulated in a state of positive pressure, so that upon puncturing the chest-wall it escapes with a distinct sound, and with force enough to blow out a lighted match. The mediastinum and heart are displaced toward the opposite side. The lung, completely atelectatic, is compressed against the spine. The pleura is inflamed, and usually covered with a fibrinous exudate which is often very abundant. A liquid effusion, sometimes sero-fibrinous, but in most cases purulent, is present. The perforating lesion can usually be discovered. It is generally in the postero-lateral region of the lung and between the third and sixth ribs. It often involves the lower part of the upper lobe. There is usually a single perforation, though one or more points of necrotic and softening pleura are not rare. When recent the actual opening is, as a rule, small and of irregular outline. In old cases the orifice may be of considerable size, and may communicate with a bronchus by a sinus through the pulmonary tissue. The opening is often valve-like. In perforation from empyema pus may ooze through the lung and escape by way of a bronchus, without the escape of air into the pleural cavity.

Symptomatology.—The onset of pneumothorax is usually sudden. It may take place during or after an effort, as during the act of vomiting, or in a paroxysm of coughing. In many cases it occurs in the absence of these causes of respiratory stress. The invasion may be unattended with special symptoms, and it occasionally happens that in pulmonary tuberculosis pneumothorax, not suspected during life, is discovered upon examination after death. More commonly, however, there is a sensation of something having given way, accompanied by pain in the upper part of the chest or in the back; urgent dyspnœa is usually present, and symptoms of shock, such as pallor, faintness, slight lividity, a rapid and feeeble pulse, and coldness of the extremities. The dyspnœa varies in intensity from a little shortness of breath, scarcely amounting to more than discomfort, to the most urgent and agonizing air-hunger. The grade of dyspnœa depends partly upon the suddenness with which air enters the pleural cavity and its amount, and partly upon the power of the sound lung to carry on the respiratory function. It depends also, in part, as Wilson Fox has pointed out, upon the condition of the blood, which in advanced states of anæmia, such as are present in late pulmonary tuberculosis, requires a diminished amount of oxygen. Sudden aphonia sometimes develops, so that the patient speaks in a whispering voice, for "want of breath."

The **physical signs** vary according as there is air only in the pleural sac, according to its volume and tension, and when, as is commonly the case, fluid also is present.

Inspection reveals enlargement and immobility of the affected side. The intercostal spaces are flat or bulging, and do not recede upon inspiration. The impulse of the heart is displaced. Distension may be absent when there is free communication with the pleura, so that the air enters and escapes without hindrance.

Palpation shows great enfeeblement, more commonly complete abolition, of the vocal fremitus.

Percussion yields resonance of tympanitic quality, which varies according to the volume of air in the cavity and the degree of tension under which it is confined. Hence every variety of tympanitic resonance may be recognized over the region of the chest occupied by air in the pleura. The sound may be vesiculo-tympanitic, as in emphysema. In this case the intrapleural tension is slight and the lung is not compressed to a high degree. It is more frequently purely tympanitic. Not rarely it is amphoric, and sometimes ringing and metallic. It may vary in pitch when there is a fistulous communication with a bronchus, being higher when the mouth is closed and lower when it is open. Under these circumstances the cracked-pot sound may in rare instances be elicited. When the tension is considerable the resonance may be very high-pitched and toneless (flat tympany), and when it is extreme resonance is lost and the percussion-sound becomes dull or even flat. Want of knowledge of the fact that air or gas in considerable volume confined in a cavity under a condition of high tension yield dulness or flatness upon percussion has frequently led to errors in diagnosis. Percussion over the dependent regions of the chest yields dulness which is frequently modified in degree by the conduction of the overlying resonance, and which rapidly changes its level with changes in the posture of the patient, in this respect differing from the condition found in ordinary pleural effusion, in which the dulness is movable to a less extent—often scarcely at all—and always much more slowly.

Auscultation affords signs that are numerous and important. The vesicular murmur is absent. If breath-sounds are heard, they are not well characterized (indeterminate) and are feeble and distant. They have also, both in inspiration and expiration, or in expiration only, an amphoric quality. The respiratory murmur upon the opposite side is often increased in intensity (puerile respiration). Over the compressed lung, near the spine, feeble bronchial breathing may be heard. Râles that are present have a metallic character. Metallic tinkling, described by Laennec as a sound like that produced by drops of water falling into a vessel (gutta cadens) or the striking of a pin on glass, is a common phenomenon. It was supposed to be caused by drops of fluid falling in the pleural cavity or to bubbles of air escaping upward through the fluid. It is probable that in pneumothorax any suddenly-produced râle, either in the bronchial tubes of the compressed lung or at the orifice of the fistula, may under certain conditions acquire this character.

The vocal resonance is amphoric. Auscultatory percussion, when practised by the ordinary method, gives rise to a sign which is noteworthy, but which when performed, as suggested by Trousseau, by striking a coin placed flat upon the chest with another coin while the ear of the auscultator is over the air-space, is striking and characteristic. The sound produced has a ringing, metallic, bell-like quality not heard upon auscultation of the chest under other circumstances.

Hippocratic succussion may be produced when fluid is present. It is a splashing sound of ringing, metallic character, heard when the body of the patient is quickly shaken or when he makes sudden movements. It may be heard only when the ear is applied to the chest, or at a distance, and the succussion may be felt both by the patient and by the hand of the observer applied to the chest.

Physical examination shows, furthermore, the signs of displacement of adjacent viscera. The heart is drawn or, in case of intrapleural pressure, pushed toward the opposite side, and the liver in right pneumothorax may be depressed to a degree not encountered in uncomplicated pleural effusion.

The **course** of pneumothorax varies greatly. Death may result at once; more frequently the fatal issue is delayed for several days or even for a period of weeks. In a considerable proportion of the cases the condition becomes chronic, and life is prolonged for months or years. Recovery often takes place in pneumothorax produced by traumatism and in that form which arises in the course of empyema, especially empyema due to infection of the pleura by the pneumococcus.

The **diagnosis** of pneumothorax does not, as a rule, present serious difficulties. The direct diagnosis depends upon the bulging of the intercostal spaces, the tympanitic percussion-sound—especially that variety of it elicited by the coin-test—and the displacement of adjacent organs. The amphoric voice and metallic tinkling are important signs. When fluid is present Hippocratic succussion has absolute diagnostic significance. When the intrapleural tension is high, percussion may yield dulness, but the coin-test yields the usual bell-like sound, and if fluid be present there is succussion. Diaphragmatic hernia may yield signs which can scarcely be distinguished from those of circumscribed basic pneumothorax, and the condition described by Leyden under the designation pyo-pneumothorax subphrenica has frequently been mistaken for true pneumothorax. The chief data in favor of the former are the history of the case as indicating previous abdominal rather than thoracic disease, the absence of cough and expectoration, slight displacement of the heart, absence of intercostal bulging, and obliteration of the area of hepatic or splenic dulness, together with the signs of downward dislocation of the liver and spleen.

A careful consideration of the history of the case, in connection with the physical signs, will serve to clear up any uncertainty as regards the differential diagnosis between pneumothorax and intra-pulmonary cavities yielding tympanitic resonance upon percussion, with amphoric phenomena, except in rare cases in which the cavity is the result of the complete excavation of the greater part or the whole of a lobe or lung. Under these circumstances the physical condition closely resembles that of pneumothorax. Nevertheless, it has been found that the bell-like quality produced by the coin-percussion is not heard over a cavity, however large; succussion is not heard, as a rule; the intercostal spaces do not bulge; and adjacent organs are not markedly displaced away from the affected side of the thorax, as in pneumothorax, but, on the contrary, are slightly displaced toward it.

Treatment.—Pain and distress in suddenly-developing pneumothorax are best relieved by the hypodermic administration of morphine, and in less acute cases the same drug by the mouth. When morphine is not well borne, codeine or cannabis Indica may be given in its place. In traumatic cases in and those following strain, in persons previously in apparently good health, spontaneous recovery has often followed without further intervention. This palliative treatment also fulfils the indications in pneumothorax supervening in the last stage of pulmonary tuberculosis. It may be supplemented, if the distress be extreme, by the cautious administration of chloroform from time to time. In any case when the signs point to high intrapleural pressure, and there is extreme dyspnœa with lividity, the excess of accumulated air may be permitted to escape by puncturing the chest with a fine needle. Cases of pneumothorax in pulmonary tuberculosis are occasionally encountered in which the general symptoms are ameliorated, and for a time, at least, no special treatment is required. When the liquid is considerable, aspiration must be performed, and in cases of hydrothorax recovery sometimes follows. Other cases are to be treated by free incision, thorough drainage, and excision of the ribs when necessary, as in ordinary chronic empyema.

HYDROTHORAX.

Definition.—An accumulation of serous fluid in the pleural cavities without pleural inflammation.

Etiology.—Hydrothorax is a secondary affection, and occurs especially in nephritis, disease of the heart, and profound anæmia. It often results from mechanical obstruction to the blood-current in the vena azygos or other intrathoracic veins in consequence of the presence of tumors or similar causes.

Morbid Anatomy.—Hydrothorax is usually bilateral in disease of the kidneys and when due to anæmia. The quantity in one pleural cavity is usually greater than that in the other. The amount varies from a few ounces to several pints. It is, however, commonly moderate. In heart-disease one pleural cavity only is usually implicated; if both, there is commonly great disparity in the amount of fluid. Hydrothorax from pressure may involve one or both sides of the chest. The fluid is always clear and the pleural membranes are smooth. The subpleural tissues may be swollen and œdematous, in consequence of the imbibition of fluid. Previous pleural adhesions may circumscribe the fluid, but as a rule it is free in the cavity. The lung is retracted in proportion to the amount of transudation. Hydrothorax is frequent in the terminal stages of visceral disease, and frequently escapes observation at the bedside.

Symptomatology.—The symptoms of the primary affection are present. When transudation takes place there is dyspnœa in proportion to the amount of the effusion and increased upon effort. This symptom frequently amounts to orthopnœa; it may be associated with cyanosis, profuse clammy sweating, lividity, and great distress and anxiety. Every effort may increase it. Pain is absent, and cough is merely an accidental or associated symptom.

Physical examination reveals the signs of unilateral or bilateral pleural effusion, without friction-sounds. In bilateral hydrothorax the mediastinum and heart are little displaced.

The **course** of hydrothorax may be very acute, even rapidly fatal. Moderate hydrothorax, even when double, may exist for a long time without giving rise to urgent symptoms. There is, however, constant danger of sudden increase. The occurrence of more or less urgent dyspnœa in disease of the heart or kidneys or in grave anæmia is frequently due to this form of pleural effusion.

Diagnosis.—The signs of effusion in one or both pleural sacs occurring in the course of cardiac or renal disease, or in anæmia or visceral cancer, with or without dropsy, and not accompanied with fever or pain, will in the greater number of cases justify the diagnosis of hydrothorax.

Treatment.—It is that of the dropsies with which hydrothorax is commonly associated. Limited ingestion of fluid, coupled with the use of saline or other purges, may be followed by disappearance of the pleural effusion, along with the general dropsy. Appropriate treatment of the primary disease is necessary. Very often hydrothorax arises as a condition of the terminal stage of the underlying disease, and is for that reason not amenable to medicinal measures the efficacy of which has already been exhausted. In such case repeated removal of the fluid by aspiration may prove palliative.

HÆMOTHORAX.

Definition.—A collection of blood in the pleural sac.

Hæmothorax is to be distinguished from hæmorrhagic pleural effusion as the result of an inflammatory process.

Etiology.—This condition is sometimes due to traumatism that causes fracture of the ribs or laceration of the lung. It may also arise from rupture of the wall of an aneurism or from any ulcerative or erosive lesion of an intrathoracic vessel. Exudative pleurisy may follow the effusion of blood.

Symptomatology.—The symptoms vary according to the quantity of blood and the rapidity of the hæmorrhage. When the volume is large, and especially when the blood is rapidly poured out, the symptoms are those of any severe hæmorrhage, such as pallor, small, thready, or imperceptible pulse, faintness, restlessness, and sighing respiration, together with dyspnœa proportionate to the mechanical interference with the respiration. Pain may be present, but it is not an invariable symptom.

The **physical signs** are merely those of an effusion of fluid into the pleural cavity. Friction-sounds are not present.

Course and Termination.—It has been experimentally shown that blood in the pleural cavity does not, in the absence of infection, give rise to inflammation. Coagulation takes place, and is followed by resorption of the serous fluid, and later of the clot. If there be coincident infection, empyema results.

A considerable proportion of traumatic cases terminate in recovery. Hæmothorax resulting from the rupture of an aneurism or the erosion of the wall of a large vessel is quickly fatal.

Diagnosis.—The signs of pleural effusion after traumatism of the chest, especially if attended by symptoms of internal hæmorrhage, justify the diagnosis of hæmothorax. These signs and symptoms in a case of thoracic aneurism or malignant disease of the œsophagus or of caries of a rib would warrant the same conclusion.

The **prognosis** depends upon the cause, and is much more favorable after injury to the chest than when the condition arises in consequence of disease of the intrathoracic vessels or of malignant disease. A rapidly formed and large hæmothorax from any cause may terminate in sudden death.

Treatment.—Hæmothorax of moderate volume resulting from traumatism should be let alone. If of large size and endangering the life of the patient by mechanical interference with respiration, an attempt may be made to draw off the serous portion by the aspirator, or the blood may be evacuated by incision. If empyema follows, it should be treated in accordance with the rules already laid down for the management of that affection.

DISEASES OF THE LUNGS.

BY FRANCIS DELAFIELD.

PNEUMONIA.

THE term Pneumonia, or Pneumonitis, is employed to designate the inflammations of the parenchyma of the lungs as distinguished from those of the bronchi and the pleura. By the parenchyma of the lung we mean the air-vesicles, the air-passages, and the smallest bronchi.

There are a number of different forms of pneumonia, distinguished from each other by their causes, their lesions, and their symptoms. In the present state of our knowledge we cannot make a scientific classification of the different forms, but have to be contented to describe the different varieties of pneumonia under arbitrary names.

We distinguish, therefore, Primary Lobar Pneumonia; Secondary Lobar Pneumonia; Lobar Pneumonia with the formation of new connective tissue; Broncho-pneumonia; Pneumonia of Heart Disease; Interstitial Pneumonia; Tubercular Pneumonia; Syphilitic Pneumonia.

PRIMARY LOBAR PNEUMONIA.

Definition.—An infectious inflammation, with exudation from the blood-vessels and the growth of pathogenic bacteria, which involves principally the air-spaces of the lungs.

SYNONYMS.—Croupous pneumonia; Fibrinous Pneumonia; Lung fever; Pneumonitis.

Etiology.—Lobar pneumonia is a very widely distributed disease. There are few countries in which it does not prevail, the mortality ranging from 1.10 to 2.30 per cent. for each 1000 inhabitants. In the United States the disease is of more frequent occurrence in the South than in the North. This the accompanying table, based on the eighth and ninth census reports, conclusively shows.

In most countries in the temperate zone the maximum frequency of the disease is from February to May.

As regards New York City, I compiled from the records of the Board of Health the deaths from pneumonia from March 1, 1871, to March 1, 1875—7873 cases. Nearly half the entire number was in children under five years of age. The smallest mortality was in persons from ten to twenty years old. The majority of the cases occurred in March, April, May, December, January, and February; the minority in June, July, and August.

In persons over five years old the curves of mortality are very regular, and the difference between the spring and winter months and the rest of the

States Wholly or in Great Part above the 39th Parallel.	Per 1000 Deaths.	Per 1000 Inhabitants.	States Wholly or in Great Part below the 39th Parallel.	Per 1000 Deaths.	Per 1000 Inhabitants.
1. Maine	51.22	0.62	1. Delaware	56.41	0.68
2. New Hampshire	62.46	0.96	2. Maryland	59.59	0.70
3. Vermont	58.58	0.59	3. Virginia, West Virginia	75.66	0.94
4. Massachusetts	56.29	0.98	4. North Carolina	71.34	0.80
5. Rhode Island	58.23	0.78	5. South Carolina	102.58	1.26
6. Connecticut	55.96	0.72	6. Georgia	99.54	1.17
7. New York	60.55	0.87	7. Florida	113.55	1.39
8. New Jersey	51.61	0.59	8. Alabama	123.81	1.47
9. Pennsylvania	44.84	0.58	9. Mississippi	127.21	1.68
10. Ohio	60.27	0.65	10. Louisana	94.15	1.75
11. Indiana	80.66	0.88	11. Texas	105.43	1.58
12. Illinois	77.94	0.96	12. Arkansas	183.42	2.98
13. Michigan	69.64	0.67	13. Kansas	112.13	1.49
14. Wisconsin	54.55	0.50	14. Kentucky	78.49	0.95
15. Minnesota	55.30	0.39	15. Tennessee	84.03	1.04
16. Iowa	75.32	0.61	16. Missouri	103.96	1.41
17. Nebraska	87.30	0.93	17. Nevada	81.30	1.18
18. Oregan	55.76	0.34	18. California	46.77	0.65
19. Colorado	50.67	0.48	19. District of Columbia	60.87	0.98
Average	61.43	0.69	Average	93.70	1.27
			Excess over Northern States	32.27	0.58

year very striking. In persons over seventy years of age the same law prevails. In children under five years of age the curves are much less regular. The curves of mortality in general correspond with those of temperature, the greatest mortality with the lowest temperature and the greatest daily range of temperature.

The disease may occur in epidemics, confined to prisons, barracks, asylums, or involving certain districts. There seems to be no question that persons living an out-of-door life in the country are less liable to the disease than are persons living in the cities.

It has always been a matter of importance to determine whether pneumonia is contagious—whether a person suffering from the disease can communicate it to others. The disease certainly occurs in circumscribed, local epidemics, and from time to time we see several persons in one house successively attacked. On the other hand, it is well known that physicians, nurses, and relatives who take care of cases of pneumonia are not often attacked by the disease. For the present the question must be considered an unsettled one.

For the production of a lobar pneumonia there must be a cause of inflammation, such as exposure to cold, and the growth of pathogenic bacteria. The organism most frequently found is that described by Fraenkel; it is said to be found in over 90 per cent. of all the cases. This same micro-organism is also found with pleurisy, pericarditis, peritonitis, and cerebro-spinal meningitis, and is present in the saliva and nasal secretions of healthy persons. Much less frequently the pneumococcus of Friedländer or the streptococci of suppuration are found.

The old conception of pneumonia was that it was simply an inflammation of the lung. Within a few years the opinion that, on the contrary, it is a general disease, of which the inflammation of the lung is the characteristic lesion, has gained very general acceptance. With our present knowledge it seems most probable that pneumonia belongs to the class of infectious inflammations; that is, it is an inflammation of the lung accompanied by the growth of pathogenic bacteria. The growth of these bacteria is attended with the formation of poisonous chemical products, and, according to the quantity and virulence of these products, the symptoms of general poisoning are more or less marked.

Children under five years of age usually have broncho-pneumonia; children between the ages of five and fifteen have either broncho-pneumonia or lobar pneumonia; adults usually have lobar pneumonia.

Morbid Anatomy.—The inflammation, as a rule, involves the whole of one lobe or the whole of one lung or portions of both lungs. Juergensen, from a study of 6666 cases, gives the following table to show the relative frequency of the situation of the lesion:

	Per cent.
Right lung	53.70
" " upper lobe	12.15
" " middle lobe	1.77
" " lower lobe	22.14
" " whole lung	9.35
Left lung	38.23
" " upper lobe	6.96
" " lower lobe	22.73
" " whole lung	8.54
Both lungs	8.07
" " both upper lobes	1.09
" " both lower lobes	3.34

The inflammation in acute lobar pneumonia is of pure exudative type, characterized by congestion, emigration of white blood-cells, diapedesis of red blood-cells, and exudation of blood-plasma, while the tissue of the lung remains unchanged. For clinical purposes it is important to have as distinct an idea as possible of the condition of the lung while it is the seat of such an exudative inflammation, so that we describe the condition in which the lung is found while the inflammation is going through its regular stages of congestion, exudation, and resolution.

During the first hours of the inflammation only irregular portions of the lobe which is to be inflamed are involved; later, the entire lobe. The lung is congested, œdematous, tough, but not consolidated. The air-spaces contain granular matter, fibrin, pus-cells, red blood-cells, and epithelial cells. The epithelium remaining on the walls of the air-spaces is swollen; there are large numbers of white blood-cells in the capillaries. The larger bronchi are congested, dry or coated with mucus; the small bronchi contain the same inflammatory products as do the air-spaces. The pulmonary pleura, as a rule, is not coated with fibrin. This is called the stage of "congestion." The stage of congestion, as a rule, only lasts a few hours, but it may be protracted for several days.

When the exudation of the inflammatory products has reached its full development, the presence of these products within the air-spaces and bronchi causes the lung to be solid, and at this time the lung is said to be in the condition of "red hepatization." The lung is now consolidated, red; its cut section looks granular, the granules corresponding to the plugs of inflammatory matter within the air-spaces. For some time after death the inflammatory products remain solid and the cut section of the lung dry, but later, with the commencement of post-mortem changes, these products soften and the cut section is covered with a grumous fluid. The air-vesicles, the air-passages, the small bronchi, and sometimes the large bronchi, are filled and distended with fibrin, pus-cells, red blood-cells, and epithelium. In spite of the pressure on the walls of the air-spaces, the blood-vessels in their walls remain pervious. The pulmonary pleura is coated with fibrin, and the interstitial connective tissue of the lung is infiltrated with the same substance. The hepatized lobe is increased in size, sometimes so much so as to compress the rest of the lung. About one-fourth of the fatal cases die in the stage of red hepatization at any time from twenty-four hours to eleven days after the initial chill.

After the air-spaces have become completely filled with the exudation, if the patient continue to live, there follows a period during which the exudate becomes first decolorized and then degenerated. This is the period of "gray hepatization." The lung remains solid; its color changes, first to a mottled red and gray, then to a uniform gray. The coloring matter is discharged from the red blood-cells and the exudate begins to degenerate and soften. The lung is found passing from red to gray hepatization at any time between the second and the eighteenth day of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. About one-half of the cases die in the condition of mottled red and gray hepatization; about one-fourth, in the condition of gray hepatization.

If the patients recover, the exudate undergoes still further degeneration and softening, and is removed by the lymphatics. This is the stage of "resolution." It should commence immediately after defervescence and be completed within a few days. But it may not begin until a number of days after defervescence, or it may be unusually protracted.

Modifications of the Inflammation.—The lung, instead of being freed from the exudate at the regular time, may remain in the condition of gray hepatization for weeks.

The quantity of inflammatory products may be so great that the blood-vessels are compressed and portions of the lung become necrotic.

There may be an excessive production of pus-cells, with infiltration of the walls of the air-spaces and of the stroma of the lung. The bronchitis may be developed in an unusual degree, and involve not only the bronchi of the inflamed lung, but also those of the other lung. The pleurisy may be unduly developed at any time in the course of the pneumonia or after it has subsided.

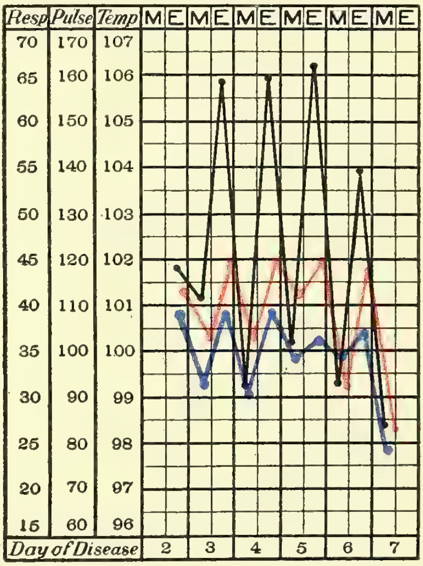
In the lobar pneumonia which accompanies epidemic influenza there is often an intense catarrhal bronchitis with a large production of muco-pus, and in some cases an excessive congestion of the lung with comparatively little hepatization.

The lymphatic vessels in the pulmonary pleura and in the septa between the lobules may be filled with pus-cells, and the pleura and the septa infiltrated with fibrin and pus.

It is often stated that lobar pneumonia can be changed into a tubercular pneumonia or a chronic pneumonia, but I believe that the cases thus described are really examples of pneumonias which were of tubercular or of productive character from the very outset.

Symptoms.—*Physical Signs.*—During the stage of congestion the lung is more dense, but is not consolidated, the bronchi and some of the air-spaces contain inflammatory products, the pleura is not yet coated with fibrin. The percussion note, therefore, remains unchanged or its pitch becomes higher, its duration shorter, and its quality less distinctly pulmonary. The respiratory murmur is either rude or diminished in intensity. The inflammatory products in the small bronchi may give a subcrepitant râle. If the larger bronchi are also inflamed, there may be coarse râles and sibilant and sonorous breathing. As there is no fibrin yet on the pleura, there is no crepitant râle. It is evident, therefore, that during this stage of a pneumonia we must expect that the physical signs will either not be very marked or else absent altogether.

Fig. 43.



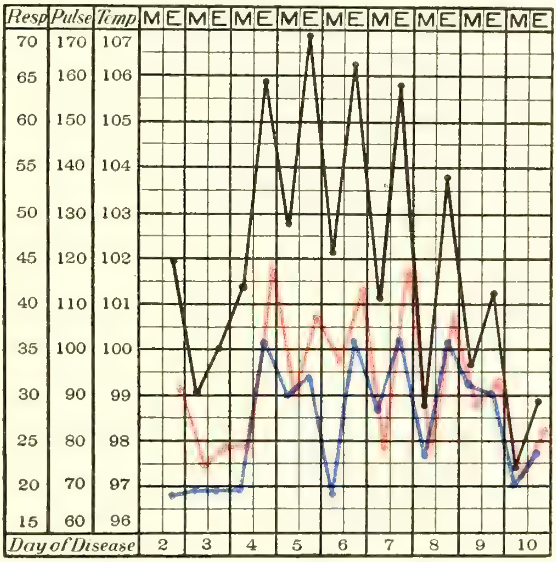
Temperature, black ; pulse, red ; respiration, blue.
Lobar Pneumonia, right lower lobe ; recovery. Expectant treatment.

During the stages of red and gray hepatization the air-spaces and small bronchi are filled with inflammatory products and impervious to air. The larger bronchi are coated with mucus or filled with fibrin. But, although in all cases the lung is consolidated, there is a good deal of difference as to the quantity of inflammatory products, the size of the consolidated lobe, the closeness with which its surface is applied to the chest-wall, and the degree of motion of which it is capable. The pulmonary pleura is coated with fibrin ; occasionally there is serum in the pleural cavity. The percussion sound, therefore, is more or less dull, or flat, or tympanitic, or of cracked-pot quality. Any considerable quantity of fluid in the pleural cavity gives flatness.

A lobe of which the air-spaces are distended with exudation, so that the lobe is increased in size and its surface pressed closely against the wall of the chest, gives either marked dulness, or flatness, or tympanitic resonance, or, in young persons, the cracked-pot sound.

The dulness on percussion is less marked, or is even absent altogether if the inflamed lobe is very much congested, but contains little exudation; if the quantity of exudation is not sufficient to distend the air-spaces and increase the size of the lobe; if the area of consolidation is small or is situated in the central portions of the lung; or if the ribs have undergone the senile changes which cause them to give increased resonance. It is especially in old persons that these reasons for the absence of dulness on percussion often exist.

FIG. 44.



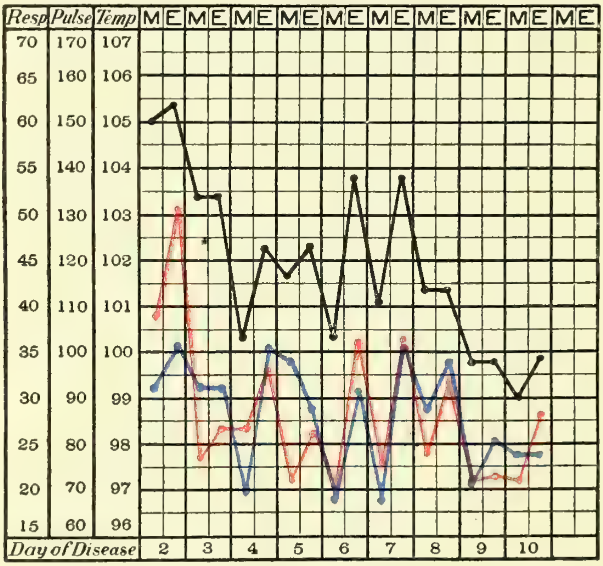
Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia, left lower and right upper lobes; recovery. No antipyretics or quinine used.

The vocal fremitus is, as a rule, increased over the consolidated lung. There is no satisfactory reason why this should not always be the case, but as exceptional conditions we find the vocal fremitus diminished or absent.

Bronchial breathing and bronchophony should be heard over the consolidated lung, but we may get bronchophony without bronchial breathing, or we may get neither bronchophony nor bronchial breathing. The absence of bronchophony seems to be due sometimes to the incomplete consolidation, sometimes to the occlusion of the large bronchi. The absence of bronchial breathing may be due to occlusion of the large bronchi or to the absence of movement of the lung. As the crepitant râle is due to the friction of the fibrin on the surface of the pleura, the râle will not be produced unless fibrin is present and the lung capable of movement. So we find in different cases a great difference as to the presence or absence of the crepitant râle. In some cases we only get it after coughing or with a forced inspiration; in some cases it is only heard at the beginning of red hepatization while the lung still moves; in some cases it is heard throughout the stages of red and gray hepatization; and in some cases it is absent altogether.

In the stage of resolution the products of inflammation are softened and rapidly absorbed, the air re-enters the small bronchi and air-spaces, the lung moves more and more freely. So with the increased motion of the lung we get the crepitant râle due to the friction of the pleura. With the softening of the inflammatory products we get the subcrepitant and coarse râles in the bronchi. The bronchial breathing and bronchophony disappear if they have been present. Normal vesicular breathing becomes more and more distinct. The percussion note loses its dull or flat or tympanitic or cracked-pot quality, and approaches nearer and nearer to the normal, but yet the changes in the percussion note last the longest of all the physical signs, and even long after complete resolution some dulness is often present.

Fig. 45.



Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia, right lower lobe; recovery. Treatment by aconitine and digitaline.

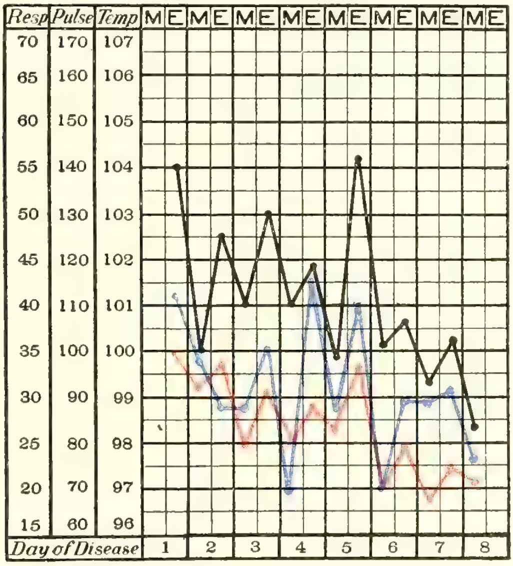
It is always to be remembered that it is in old persons that the physical signs are the least constant and the least well marked.

Rational Symptoms.—In from one-sixth to one-third of the cases there are prodromic symptoms. Chilliness, a little fever, general malaise, and feelings of oppression about the chest continue for from one to four days. These symptoms probably correspond to a protracted period of congestion.

In about 90 per cent. of the cases there are, during the first twenty-four hours, one or more decided chills, and it is from the time of the chill that we count the days of the disease.

The temperature rises at once, and reaches its maximum by the afternoon of the first, second, or third day (See Fig. 45), but very often the highest temperature of the disease will be reached during the twenty-four hours preceding defervescence. An afternoon temperature of 104° and a morning temperature of 102° or 103° F. are about the normal temperatures of a lobar pneumonia. A sudden rise of temperature during the course of the disease indicates the extension of the pneumonia or the development of a complication. The removal of the patient from one house to another is, as a rule, followed by a rise of temperature, and in persons not suffering from malarial poisoning and not taking antipyretics there are quite often irregular rises and falls of several degrees of temperature which we cannot account for. Pneumonias involving the upper lobes usually have high temperatures. The height of the temperature is usually in proportion to the severity of the disease, but patients may get worse with a falling temperature, or may die with temperatures below the normal, or, very rarely, have no rise of temperature throughout the disease. The accompanying charts show variations in the ratio of temperature, pulse, and respiration in various conditions and under different plans of treatment.

Fig. 46.



Temperature, black : pulse, red ; respiration, blue.
Lobar Pneumonia, right lower lobe ; recovery. Treatment with aconitine and digitaline.

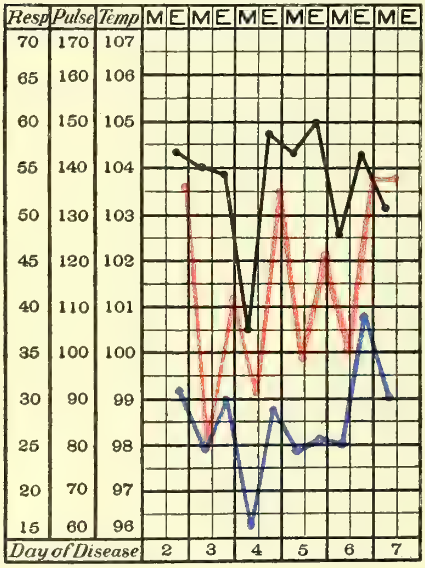
Defervescence may take place at any time from the second to the eighteenth day of the disease. It occurs most frequently on the seventh day ; next on the fifth, eighth, sixth, and ninth days, in order. The fall of temperature usually begins in the evening, and within from six to forty-eight hours it reaches the normal or for a time falls below it. Occasionally a rapid defervescence with a fall of temperature to 97° or 96° F. is attended with so much prostration and such a feeble heart-action that the condition of the patient is alarming. It is said that epistaxis, hæmaturia, or hæmorrhage from the bowels may accompany defervescence.

In the pneumonias which complicate epidemic influenza there may be no marked defervescence, but a gradual fall of temperature extending over many days, and in some cases the fever persists after the pneumonia has resolved.

A rise of temperature after several days of partial or complete defervescence usually means pleurisy or empyema, but it may indicate a fresh pneumonia, abscess of the lung, or gangrene of the lung.

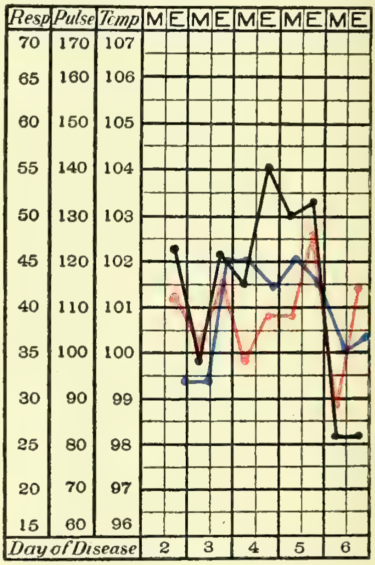
The condition of the heart's action and that of the pulse are of great importance. In a favorable case the pulse ought to be about 100 to the minute, and fairly full. A pulse of over 120 is always cause for anxiety. The liability of failure of the heart's action, either gradual or sudden, constitutes one of the greatest dangers of pneumonia. It is not certain what the cause of the heart failure is, but it seems probable that it is due to the effects of the poison produced by the pathogenic bacteria of the disease.

Fig. 47.



Temperature, black; pulse, red; respiration, blue. Lobar Pneumonia, right lower lobe; death. Complicated by acute gastro-enteritis and by peritonitis.

Fig. 48.



Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia, right upper lobe; recovery. Expectant treatment.

The character of the heart failure resembles that of diphtheria: either the pulse gradually becomes more rapid and feeble or there is a sudden cessation of the heart's action. The liability to heart failure seems ordinarily to begin on the third day of the pneumonia, and to be greatest on the day before defervescence.

In persons already suffering from chronic endocarditis the danger of heart failure is very great; and it must be remembered that an old aortic or mitral stenosis may exist without a murmur.

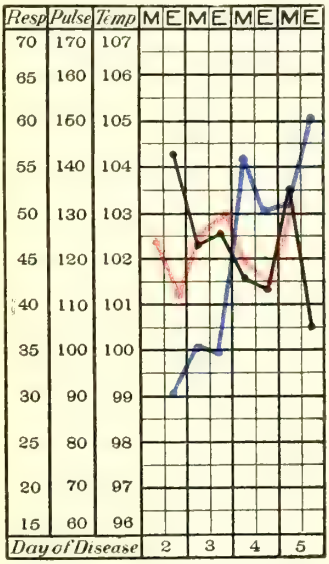
A complicating pericarditis, as a rule, increases the frequency of the pulse.

The breathing during the invasion of the disease is rapid and oppressed. As the disease progresses the character of the breathing varies with the severity of the case. Rapid, labored, and insufficient breathing indicates either inflammation of a large part of the lung, excessive congestion of the lung, an intense general bronchitis, failure of the heart's action, or an intense pleurisy or pericarditis.

Cough may be developed as one of the first symptoms, or come on at any time in the disease, or be deferred until resolution has commenced. In old persons the cough is often slight or absent altogether.

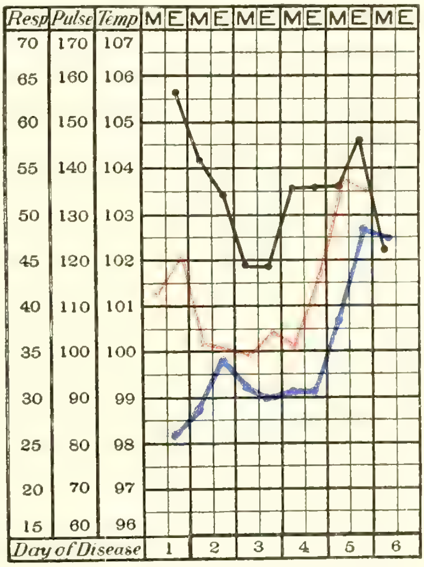
The characteristic sputa of pneumonia are little rounded, viscid pellets of red, yellow, or brownish color, mixed with thin, fluid mucus, the so-called rusty sputa. In rare cases, with a severe invasion, the patients may cough up a little pure blood at the beginning of the disease. In the bad cases the sputa are changed and the patients cough up considerable quantities of thin, dark-colored fluid. In some cases throughout the disease there will be nothing but a little white mucus coughed up from time to time. In cases with an excessive catarrhal bronchitis the quantity of expectoration may be very large and like that of an ordinary bronchitis. It is by no means unusual, especially in old people, for the expectoration to be absent altogether.

FIG. 49.



Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia, whole of right lung; death.

FIG. 50.



Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia complicating Influenza, left lower lobe; general bronchitis; death.

Pain over the inflamed lung, referred to the region below the nipple, is developed within twelve hours after the initial chill in the majority of cases, and after three or four days gradually disappears. This pain is sometimes so intense as to be for a time the principal symptom. In some persons, however, there is no pain until resolution commences and the cough becomes troublesome. In old persons there is often not only an absence of pain, but there are no abnormal sensations whatever in the chest.

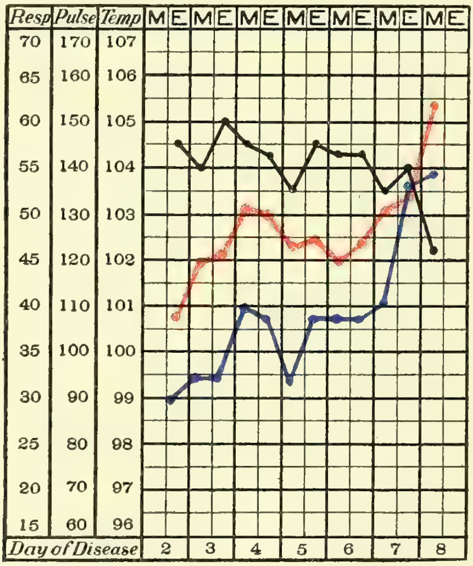
The appearance of the face is characteristic. There is a deep-red flush at about the centre of each cheek, and the expression of the face is a curious mixture of anxiety and apathy.

The skin may be either hot or dry or bathed in perspiration throughout the disease.

The tongue is coated with a white fur, but in the favorable cases remains moist. A dry tongue indicates a severe form of the disease.

Headache, restlessness, and sleeplessness are troublesome during the first days of the disease in many of the patients. Delirium and stupor belong to the severe cases. The alcoholic patients often have an active delirium or delirium tremens. Old persons often become apathetic or mildly delirious.

FIG. 51.



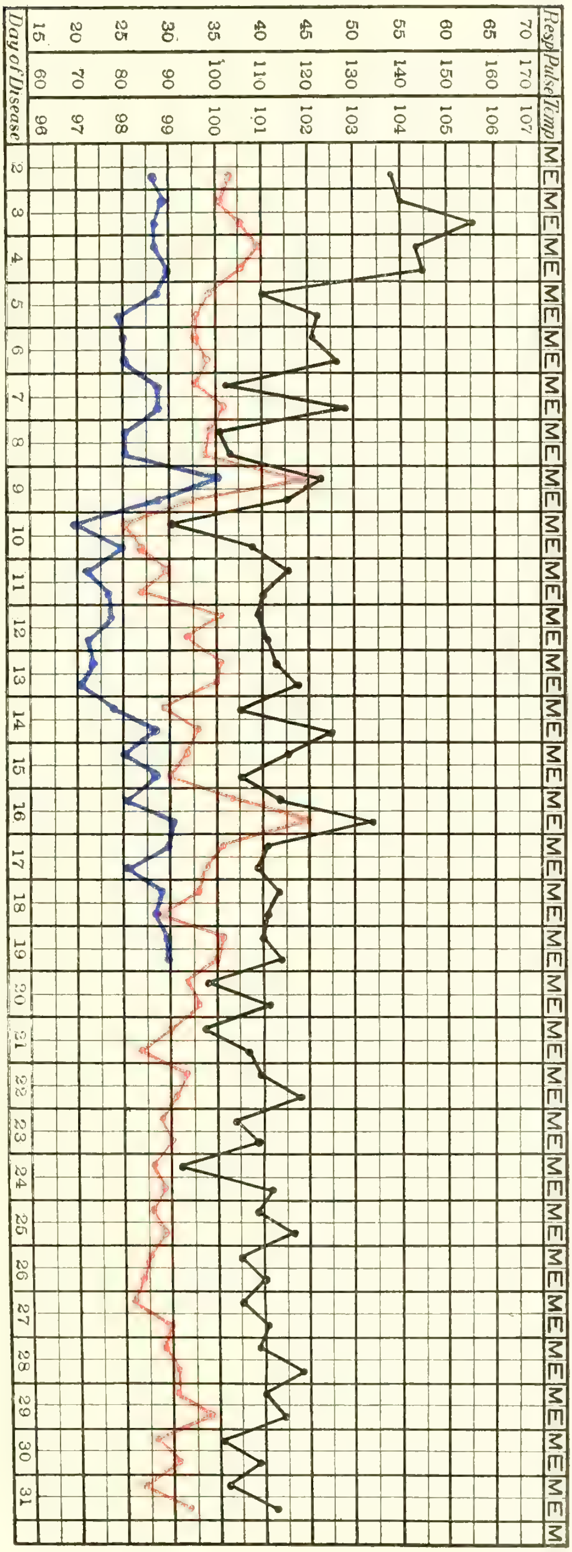
Temperature, black; pulse, red; respiration, blue.
Lobar Pneumonia, entire left lung; death.

The invasion of the disease is often attended with vomiting, less frequently with diarrhœa.

The urine is diminished in quantity and high-colored. It often contains a little albumin and a few casts, due to acute degeneration of the kidney. It is said that sodium chloride and some other inorganic salts are diminished in quantity.

Complications.—At any time in the course of a pneumonia or after

Fig. 52.



Temperature, black; pulse, red; respiration, blue.

Lobar Pneumonia complicating Influenza. Left lower lobe hepatized, resolution complete on sixteenth day. Right lower lobe hepatized on sixteenth day, resolution on twenty-fourth day. Left pleural cavity one-fourth full of serum on thirty-first day. Temperature did not fall to the normal until the fifty-second day.

defervescence there may be developed pleurisy with effusion or empyema on the same side as the pneumonia. The ordinary course is for the patient to get to the sixth or seventh day of his pneumonia and behave as if he were about to get well, but yet without complete defervescence. After a few days the temperature rises a little with pleurisy with effusion, a good deal with empyema. The physical signs are those of fluid in the pleural cavity, but very often bronchial breathing and bronchophony are heard below the level of the fluid.

A catarrhal bronchitis involving the larger bronchi of both lungs is not infrequent, especially with the pneumonia of epidemic influenza. The patients cough up large quantities of mucus, often mixed with blood. Coarse râles and sibilant and sonorous breathing can often be heard over both lungs. The dyspnœa is more troublesome, the temperature higher, and the tendency to heart failure and venous congestion more decided. (See Fig. 50.)

Acute pericarditis is a serious complication. It may be that there will be nothing to call attention to the condition of the heart, and the case seems only to be a pneumonia of severe type; or there may be decided præcordial pain, a rapid and feeble pulse, a pericardial friction sound, rapid breathing, and cyanosis. It occasionally happens that the symptoms of the pericarditis are more marked than those of the pneumonia, so that it is even possible for the pneumonia to be overlooked.

A previously-existing chronic endocarditis adds much to the dangers of pneumonia. The heart's action is likely to be disturbed and the condition of general venous congestion established. It must not be forgotten in such cases that a well-marked mitral or aortic stenosis may give no murmur at all.

Acute meningitis is an infrequent complication, but a very fatal one. It may run its course without giving any distinctive symptoms, or the delirium may be more active, with contractions of groups of muscles or general convulsions.

Jaundice is seen both in mild and in severe cases. It seems to be a non-obstructive jaundice without symptoms. Acute degeneration of the kidneys of mild type is of ordinary occurrence. It seems to do the patients no harm, and to give rise to no symptoms except the presence of albumin and casts in the urine.

Acute exudative nephritis is of much less frequent occurrence. It is not likely to prove fatal of itself, but it may add to the dangers of the pneumonia.

Persons already suffering from chronic nephritis are very unfavorable subjects for an attack of pneumonia; not many of them recover. Quite often the chronic nephritis is one which has given no symptoms until the time of the pneumonia.

The Course of the Disease.—1. *The Regular Cases.*—These cases may be mild or severe, they may terminate in recovery or in death, but they all have this in common, that the clinical picture is that of an inflammation of the lung with comparatively little evidence of general poisoning. The disease begins with the chills, rapid rise of temperature, sleeplessness and restlessness, vomiting, pain in the side, cough, expectoration, and dyspnœa. These symptoms continue either mildly or severely; after the third day comes the liability to heart failure, and finally at the regular times comes defervescence or death. The severity of the symptoms is directly in proportion to the extent of lung involved and to the intensity of the inflammation.

2. *The Infectious Cases.*—In these cases the symptoms have no necessary relation with the extent of lung involved; in many of them only a part of one lobe is inflamed. The patients behave as if they were poisoned. Their prostration is marked, the emaciation rapid. The temperatures are high, the heart's action is rapid and feeble, the tongue is dry, the cerebral symptoms are marked, and the disease is very usually fatal.

3. The rational symptoms run their regular course, but the physical signs are slow in developing, so that it may be as much as eight days before they are really well marked.

4. The inflammation, instead of remaining confined to the lobe in which it began, may extend to other portions of the lungs. Each extension of the inflammation is attended with an exacerbation of the symptoms.

5. There are rare cases in which nearly the whole of both lungs becomes at once inflamed; the interference with breathing is overwhelming, and death results very soon.

6. Resolution, instead of beginning within one or two days after defervescence, as it should, may be delayed for from one to ten weeks. And yet even after these long periods the inflammatory products may be absorbed.

7. In some cases, not necessarily belonging to either the infectious or the alcoholic classes, the delirium is an unusually marked symptom. In some of these patients the delirium continues for some days after defervescence or even after resolution is completed. In a few cases the delirium is succeeded by permanent insanity.

8. Persons already suffering from chronic alcoholism, if they have an attack of lobar pneumonia, are likely to have high temperature, active delirium, or delirium tremens.

9. The pneumonia of old persons often runs an irregular course. The extent of lung inflamed may be small and the physical signs uncertain—a little dulness on percussion, a few subcrepitant râles, a diminished intensity of breathing, or even no physical signs at all. The patients usually have chilliness or a distinct chill to mark the invasion of the disease, and more or less fever while it is running its course. The appetite is lost, and there may be nausea and vomiting. The pulse is rapid and often feeble. Either stupor or a mild delirium is often present. The prostration is out of proportion to the extent of lung inflamed. The characteristic cough, expectoration, and pain in the chest are absent altogether or imperfectly developed; even the breathing may not be at all changed. The disease is, however, very fatal in old persons, and some of them die quite suddenly after seeming to be only moderately sick for a few days.

10. The pneumonia which accompanies epidemic influenza has, in New York, presented certain peculiarities. In many cases there was an intense catarrhal inflammation of the larger bronchi or both lungs, with profuse expectoration of mucus and more or less blood. Some of the fatal cases showed very marked congestion of the inflamed lung, with comparatively little consolidation; and this corresponded with an imperfect development of the physical signs of consolidation during the patient's life. Very often there was no regular defervescence, but a slow fall of temperature extending over a number of days, and sometimes not reaching the normal until after resolution was completed. In some cases the whole duration of the disease was unusually long, and defervescence and resolution did not take place until after three or four weeks (See Fig. 52). Failure of the heart's action, with venous congestion of the lungs and other viscera, was often present. The pneumonia was followed by empyema in an unusually large number of cases.

11. The course of the disease is changed by the complicating lesions—meningitis, bronchitis, pleurisy, pericarditis, endocarditis, or nephritis.

Modes of Death.—The patients die with heart failure; from the extent of the inflammation; from general poisoning; from one of the complications; or from thrombosis of the coronary arteries.

Duration.—In the cases which recover defervescence takes place at any time from the second to the thirty-second day, most frequently on the seventh or fifth. Resolution is accomplished within a few days after this, but may be delayed up to ten weeks.

In the fatal cases death may take place at any time within the limits of five hours and thirty days—most frequently on the seventh, eighth, and tenth days. In persons over seventy years old death is most common on the fifth, sixth, and seventh days.

The mortality from pneumonia is a considerable one, ranging in hospitals from 12 to 34 per cent. It seems to be the general impression that the disease is more fatal now than it was a number of years ago, but it is difficult to determine this accurately. A study of this subject has been made by Drs. Townsend and Coolidge, who have worked up the records of the Massachusetts General Hospital from 1822 to 1889. They arrived at the following conclusions:

1. In the one thousand cases of acute lobar pneumonia treated at the Massachusetts General Hospital from 1822 to 1889 there was a mortality of 25 per cent.

2. The mortality has gradually increased from 10 per cent. in the first decade to 28 in the present decade.

3. This increase is deceptive, for the following reasons, all of which were shown to be causative of a large mortality:

(*a*) The average age of the patients has been increasing from the first to the last decade.

(*b*) The relative number of complicated and delicate cases has increased.

(*c*) The relative number of intemperate cases has increased.

(*d*) The relative number of foreigners has increased.

4. These causes are sufficient to explain the entire rise in the mortality.

5. Treatment, which was heroic before 1850, transitional between 1850 and 1860, and expectant and sustaining since 1860, has not, therefore, influenced the mortality-rate.

6. Treatment has not influenced the duration of the disease or of its convalescence.

Treatment.—Lobar pneumonia is a disease for which there is no routine treatment applicable to every case; on the contrary, much judgment is required to decide what is the best way of managing each patient.

As a rule, all the patients are to be kept in bed and on a fluid diet until the temperature has fallen to the normal and the exudate has disappeared from the lung. But there are cases in which, even while the inflammation is active, a little scraped beef improves the heart-action; and in patients in whom defervescence or resolution is delayed for an unusually long time it may be wise to give solid food and to allow the patient to sit up before the usual time.

The headache, sleeplessness, and general discomfort may require the use of opium, sulphonal, the bromides, or chloral.

For the excessive pain in the side large poultices, repeated every two hours, and hypodermics of morphine are to be used.

For the feeble heart we employ alcoholic stimulants, digitalis, caffeine, or strophanthus. A very good combination is 5 grains of potassium iodide, 1 minim of fluid extract of digitalis, and 20 minims of fluid extract of convallaria, given together every three hours.

When there is excessive congestion of the lungs, with great dyspnœa and many coarse and subcrepitant râles all over the lungs, relief can be obtained by repeated dry cupping, by hypodermic injections of one-fiftieth of a grain of nitro-glycerin, or by 20-drop doses of tincture of nux vomica.

If there is a good deal of general catarrhal bronchitis, the patients can be benefited by dry cupping or by the use of one-tenth of a grain of ipecac every hour.

For the exudative inflammation of the lungs the plans of treatment most frequently adopted are:

Venesection employed once or repeated several times, the quantity of blood taken to be considerable. This plan is not often employed at the present time.

Large doses of calomel, 12 to 30 grains, placed dry on the tongue—from one to four such doses. This plan seems to answer well for some cases, to be of no use in others, and is attended with risk of producing salivation.

Small doses of calomel, $\frac{1}{4}$ to 1 grain given every hour up to six doses. This seems to be of moderate efficacy.

Drachm-doses of magnesium sulphate, given every hour up to eight doses. This, again, does not seem to be of much value.

The employment of either tincture of aconite or tincture of veratrum viride, in doses of from 2 to 5 drops, at first every hour and later at longer intervals, has given good results in the hands of some physicians.

Cold affusions to the chest, cold baths, blisters, the antipyretic drugs, quinine, and carbonate of ammonium have all been much used, but are all of doubtful efficacy.

The treatment of the exudative inflammation which seems to me the most satisfactory is that by the combined use of aconitine, digitaline, and whiskey. The aconitine and digitaline used are those made by Merck. The dose is $\frac{1}{6}$ m. gm. (gr. $\frac{1}{400}$) of aconitine and $\frac{1}{8}$ m. gm. (gr. $\frac{1}{500}$) of digitaline in tablets; the two drugs are given together. The frequency of the dosing is regulated by the pulse. We endeavor to get a pulse of about 80 and of good quality. In order to do this it may be necessary to give the drug every fifteen minutes, every half hour, every hour, or every two hours, as the case may be. If the pulse be slow enough, but too feebie, whiskey must be given at regular intervals. It is not safe to follow this plan of treatment unless the patient can be watched. If it be carried too far, the pulse may fall to 30 and the patient pass into a condition approaching collapse. This plan of treatment does not seem to be of any service in the cases in which the symptoms of poisoning are out of proportion to the extent of lung inflamed, nor in the cases which are complicated by pericarditis. Such patients are not benefited by any treatment, and are very apt to die.

It is for the patients of this class that the experiments which have been made with the inoculation of the blood-serum of persons who have passed the crisis of a pneumonia hold out some promise. The theory is, that the blood-serum of such persons contains an antitoxic substance capable of neutralizing the poison produced by the growth of the pneumococci.

SECONDARY LOBAR PNEUMONIA.

Definition.—An exudative inflammation involving one or more lobes of one or both lungs, occurring in persons already suffering from some disease or injury.

Etiology.—Persons who are confined to bed by an infectious disease, by injuries or inflammation of the brain and spinal cord, by surgical operations, or by severe injuries are liable to have venous congestion of the dependent portions of the lung and to inhale substances and streptococci which can irritate the lung. In this way they often contract either a true broncho-pneumonia or a pneumonia which somewhat resembles a lobar pneumonia.

Morbid Anatomy.—The inflammatory process involves irregular areas of one or of both lungs. We find these areas after death in the condition of red or gray hepatization and surrounded by congested lung, but no complete consolidation of an entire lobe. The inflammation is of exudative type, with fibrin, pus, epithelium, and streptococci in the air-spaces and small bronchi.

Symptoms.—In many cases the pneumonia can hardly be said to give either rational symptoms or physical signs. We find the lesion after death, but are not certain of its existence during life. In some cases, however, there are chills, fever, rapid breathing, pain, cough, and expectoration, with the physical signs of bronchitis or of consolidation of small portions of the lung.

The **treatment** of such a pneumonia is unsatisfactory.

LOBAR PNEUMONIA, WITH THE FORMATION OF CONNECTIVE TISSUE.

It is well known that in some forms of inflammation of the lung there is a production of new connective tissue around the bronchi and blood-vessels, in the septa between the lobules, and in the walls of the air-spaces. It is not so well known that in these same forms of pneumonia there may be also a production of new connective tissue in the cavities of the air-spaces and of the small bronchi. This new tissue either grows directly from the walls of the air-spaces or is formed out of plugs of coagulated matter and of cells which are formed within their cavities.

Such a productive pneumonia has been recognized under a variety of names—gray induration, fibroid induration, cirrhosis, interstitial pneumonia, chronic pneumonia, desquamative pneumonia, parenchymatous pneumonia, etc. If we look over all the different lungs in which such a productive pneumonia has been developed, we find that they can be classified as follows:

1. A productive pneumonia associated with the growth of tubercle bacilli.
2. A productive pneumonia associated with the growth of actinomyces.
3. A productive pneumonia due to the inhalation of particles of coal or of stone.
4. A productive pneumonia caused by constitutional syphilis.
5. A productive pneumonia secondary to changes in the pleura.
6. Broncho-pneumonia.
7. A special form of lobar pneumonia.

It is concerning this last variety of productive pneumonia that our information is the least exact, and it is to this variety that I wish especially to call attention. The ordinary belief has been that it is possible for a regular exudative lobar pneumonia, instead of resolving, to be succeeded by a chronic productive inflammation. I believe, on the contrary, that a regular exudative lobar pneumonia terminates only in resolution or in death, and that lobar pneumonia with the production of new connective tissue is from the first a special form of inflammation of the lung. My reason for this belief is that I have seen a number of lungs which seem to show the different stages of the inflammatory process.

The literature on the subject is not very abundant. Charcot[1] describes this condition as following one or more attacks of ordinary lobar pneumonia. Coupland[2] gives a very good description with drawings, and believes that the new tissue is formed from the intra-alveolar exudation of ordinary lobar pneumonia. Kidd[3] describes two cases with a subacute history which he regards as cases of lobar pneumonia terminating in induration. Buhl[1] considers the disease to be a primary one, which runs a subacute course and has nothing to do with ordinary pneumonia. Heitler[2] gives an account of the disease as observed in five cases. The development of the disease, he says, is more or less acute, with fever, dyspnœa, cough, prostration, sometimes rigors; the constitutional depression is much less marked than in acute pneumonia. The fever is irregular, and not over 102° F. The sputum is mucous, muco-purulent, or fetid. In two of the cases there was retraction of the wall of the thorax. The consolidation involved in three cases the right upper lobe, in one case the whole right lung, in one case the lower right lobe. The hepatization was smooth, with necrotic and cheesy areas and cavities. The course of the disease was subacute, but with an acute invasion, lasting from fifty days to nine months and nineteen days. Wagner[3] has described six cases apparently belonging to this group, running a subacute course with retraction of the wall of the chest, but terminating in recovery.

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[1] *Rev. Mens. de Méd. et Chir.*, 1878, p. 776.

[2] *Trans. Lond. Path. Soc.*, vol. xxx. p. 224.

[3] *Lancet*, April 5, 1890.

I have seen twelve cases which seem to belong to this group, and to demonstrate that there is a form of lobar pneumonia which is, from the outset, anatomically distinct from the ordinary form. It is from the first an exudative inflammation with the production of new tissue, not a simple exudative inflammation. Such an inflammation naturally lasts longer and is more likely to become chronic than is the case with a simple exudative inflammation. I can see no reason to believe that in ordinary lobar pneumonia the pus and fibrin are ever replaced by connective tissue.

The development of the lesion seems to be as follows:

1. Congestion of the lung; exudation of serum, fibrin, and pus into some of the air-spaces; the formation in other air-spaces of irregular plugs with prolongations from one space into others, the plugs composed of a nearly homogeneous or finely-fibrillated material, none of them large enough to fill or distend the air-spaces; a swelling and thickening of the walls of the air-spaces, with a very considerable increase in the number of epithelial cells which cover them; more or less general catarrhal bronchitis; fibrin on the pulmonary pleura.

2. New cells, of the type of connective-tissue cells, are formed in the plugs; the walls of the air-spaces are more swollen, and may be infiltrated with small round cells; new blood-vessels are formed in the plugs which can be artificially injected from the pulmonary vessels. The gross appearance of the lung at this time is usually characteristic. One or more lobes are consolidated; they are not large as in ordinary pneumonia; their color is red or gray; the cut surface is smooth, not granular.

3. The growth of new connective tissue within the air-spaces, in their walls, and along the arteries and bronchi is so extensive that many of the air-spaces are obliterated. The surface of the lung is now covered with connective-tissue adhesions; the bronchi contain muco-pus; the lung is red,

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[1] Buhl, *Briefe*, p. 47.

[2] *Wiener med. Wochenschrift*, 1884 and 1886.

[3] *Deutsch. Arch. f. klin. Med.*, vol. xxxiii. V. *Kahlden Ziegler's* Beiträge, xiii. p. 279.

mottled with white or gray or black; it is dense and hard; portions of it may be necrotic or cheesy or broken down into cavities.

Four of my cases illustrate the first period of the development of the lesions:

CASE 1. The duration of the disease was ten days. The entire left lung was consolidated, small, smooth, of gray color, with fibrin coating the pulmonary pleura. The right lower lobe was partly hepatized and red. The walls of the air-spaces were thickened and coated with epithelial cells. There was a growth of new connective tissue around the blood-vessels and bronchi. The air-spaces contained small anastomosing plugs of a nearly homogeneous matter.

CASE 2. The duration of the disease was seventeen days. The left lower lobe was consolidated, small, red and smooth, its surface covered with old adhesions. The walls of the air-spaces were thickened: their cavities contained the plugs already described.

CASE 3. The duration of the disease was fourteen days. There was a general bronchitis. The right upper lobe was consolidated, of reddish-gray color, its pleura coated with fibrin. The walls of the small bronchi were thickened and infiltrated with cells. The walls of the air-spaces were thickened and coated with epithelium. Some of the air-spaces contained pus and fibrin, others the plugs already mentioned.

CASE 4. The duration of the disease was seven days. The left lower lobe presented the regular picture of the red hepatization of ordinary pneumonia. The left upper lobe was consolidated, small, smooth, and red. The walls of the air-spaces were thickened and coated with epithelium; their cavities contained pus and fibrin or the plugs.

Six of my cases illustrate the second period of the development of the lesion:

CASE 5. Duration, nineteen days. The right upper lobe was consolidated, smooth, and red. There was a growth of new connective tissue around the arteries, in the septa, and in the walls of the air-spaces. Some of the air-spaces contained epithelium, others plugs of the same shape and appearance as seen in the preceding group of cases; but there were, in addition, connective-tissue cells imbedded in the basement-substance composing the plugs.

CASE 6. Duration, sixteen days. The right middle and lower and left lower lobes were consolidated, small, and smooth. The walls of the air-spaces were thickened. Some of their cavities contained fibrin and pus, others plugs of connective tissue containing blood-vessels.

CASE 7. Duration, twenty-three days. The left lower lobe was consolidated, small, and gray. The walls of the air-spaces were thickened; they contained plugs of connective tissue.

CASE 8. Duration, twenty-eight days. The left upper lobe was consolidated, small, smooth, and black. The walls of the air-spaces were thickened; they were covered with epithelium and contained plugs of connective tissue.

CASE 9. Duration, six days. General bronchitis. The left upper lobe was consolidated, large, and red. The walls of the air-spaces were thickened. Some contained pus and fibrin, others plugs of connective tissue.

CASE 10. Duration, thirteen days. The left lower lobe was in the condition of ordinary red hepatization. The right upper lobe looked like the resolution of an ordinary pneumonia, and some of the air-spaces contained degenerated exudation, but in others there were plugs of connective tissue.

Two cases illustrate the third period of development of the lesion :

CASE 11. Duration, fifty-two days. The left lung was covered with old adhesions, consolidated, hard, smooth, mottled red and white, small. The growth of new connective tissue in the walls of the air-spaces and in their cavities had nearly obliterated the natural structure of the lung.

CASE 12. Duration, fifty-one days. The pleura was thickened and coated with fibrin. The left pleural cavity was half full of serum. The left upper lobe was consolidated and of a pinkish-white color. It was almost entirely changed into connective tissue.

Etiology.—In three of the cases there was a distinct history of prolonged exposure to cold and wet. In one case the symptoms followed immediately after the patient's having fallen into an excavation. In one case for twenty days before the initial chill the patient was miserable and had a troublesome cough. In one case for twenty-one days before the initial chill the patient suffered from headache, loss of appetite, and prostration. In one case, under observation throughout, there was an attack of lobar pneumonia terminating in resolution after eleven days ; the patient was discharged from the hospital well, and after an interval of eighteen days came the beginning of the fatal attack in the other lobe of the same lung. In one case the patient stated that he had an attack of pneumonia five years before, and that for one year he had been troubled with cough and muco-purulent expectoration.

Symptoms.—In ten of the cases the invasion of the disease was marked by chills and a rapid rise of temperature. There was cough in all the cases, the sputa rusty in five cases, muco-purulent in two cases, bloody in one case. The temperature was rarely over 104° F., and in some of the cases not over 100° F. In some of the cases delirium is noted as a prominent symptom. One case was supposed to be acute phthisis, one acute meningitis, and one acute general tuberculosis. Three of the patients passed fairly into the typhoid condition. The physical signs of the consolidation of the lung were well marked, except in one case. The duration of the disease was for six, seven, ten, thirteen, fourteen, sixteen, seventeen, nineteen, twenty-three, twenty-eight, fifty-one, and fifty-two days—in most of the cases longer than that of an ordinary lobar pneumonia.

There seems to be, therefore, a form of lobar pneumonia which is anatomically different from the regular form. Its physical signs are, of course, the same, but its clinical symptoms are somewhat different. Although the patients have the same chill, fever, cough, expectoration, and pain as in the regular cases, yet there is something about the course of the disease which makes its diagnosis possible even during its early days. The temperature does not run high, but the tendency to cerebral symptoms and the typhoid state is very marked, and most of the cases run a protracted and subacute course. Concerning the prognosis it is not possible to speak certainly, but there seems no reason why recovery should not be possible.

BRONCHO-PNEUMONIA.

Definition.—An infectious inflammation, with exudation from the blood-vessels, a formation of new connective tissue, and the growth of pathogenic bacteria, which involves principally the walls of the bronchi and those of the air-spaces which surround the inflamed bronchi.

SYNONYMS.—Capillary bronchitis; Lobular pneumonia; Catarrhal pneumonia.

There seems to be no form of pneumonia which does not have associated with it more or less bronchitis, so that every pneumonia is in one sense a broncho-pneumonia. But it has long been recognized that there is one form of inflammation of the lung which is different from others, and in which the share of the bronchi is especially important. For several reasons, however, the popular notions concerning the disease have become somewhat confused. It was seen that in some cases, while there was a bronchitis extending down to the small bronchi, there was no consolidation of the lung, and so these were called cases of capillary bronchitis. It was seen that there were cases of general bronchitis with consolidation of circumscribed portions of the lung, and it was inferred that the inflammation extended from the bronchi to the air-spaces which empty into them; so these were called cases of lobular pneumonia. This, however, was an error in observation. Areas of atelectasis do correspond to bronchi, but the areas of consolidation do not so correspond. It was seen that in some cases the symptoms and lesions could not be distinguished from those of phthisis, and it was not understood that the only real difference between the two was the presence or absence of the tubercle bacilli. Numbers of curious ideas were connected with the term "catarrhal inflammation," and it was not realized that a catarrhal inflammation is nothing but an exudative inflammation occurring in a mucous membrane.

It is to be regretted that the erroneous idea still exists that a broncho-pneumonia is simply an inflammation of the bronchi which extends to the air-spaces opening into these bronchi.

Etiology.—Broncho-pneumonia is the ordinary pneumonia of children; it is frequently seen in young persons, and occasionally in adults and old persons.

It occurs as a primary inflammation, is often secondary to measles, whooping cough, and diphtheria, less frequently to the other infectious diseases. Persons confined to bed by injury or disease and persons with emphysema are liable to subacute forms of broncho-pneumonia.

The disease is most frequent during the cold and wet months of the year; in some cases there is a history of exposure to cold; in others no exciting cause is discoverable. Children crowded together in asylums are especially liable to the disease. The same patient not infrequently suffers from several attacks of broncho-pneumonia.

The pathogenic bacteria are either the pneumococci of lobar pneumonia or the streptococci of suppuration.

Morbid Anatomy.—In persons who die of broncho-pneumonia the lungs after death present a considerable variety in their gross appearance. The mucous membrane of the trachea and large bronch may appear normal, or is congested and coated with mucus, or the small bronchi may contain pus. The walls of the small bronchi are thickened, so that a section of the lung looks studded with little nodules. In some cases most of the small bronchi of both lungs have their walls thickened in this way; more frequently it is only the bronchi of one lung or of one lobe. Around the bronchi whose walls are thickened are zones of consolidated lung varying in size from that of a pin's head to that of a pea. Scattered through one or both lungs are irregular areas of consolidation having no definite relation with the bronchi. They may be so large and numerous that an entire lobe or an entire lung is completely consolidated. The pulmonary pleura may be coated with fibrin. In the lungs of very young children there may be consolidated shrunken portions due to collapse of the air-vesicles, the so-called areas of atelectasis. The same condition is found with bronchitis and in children who die so soon after birth that the whole of the lungs have not become aërated. The bronchial glands are usually swollen and inflamed. The smaller bronchi may be dilated. In the portions of lung which are not consolidated the air-spaces may be somewhat dilated. Occasionally some of the air-spaces are ruptured and the septa between the lobules are infiltrated with air.

In order to understand the true anatomical characters of broncho-pneumonia it is necessary to determine which of these different changes is essential and constant and which are accessory and inconstant.

The essential and constant lesion is a productive inflammation of the walls (not the mucous membrane) of the bronchi and of the air-spaces immediately surrounding the inflamed bronchi. The walls of the bronchi are thickened and infiltrated by a growth of new cells; the walls of the air-spaces are thickened; their cavities are filled with new connective tissue, or with fibrin, pus, and epithelium. The inflammation is from the first not exudative, but productive; that is, with the formation of new tissue. It involves the medium-sized and smaller bronchi of both lungs, but is not everywhere equally severe; in some parts of the lungs the lesions are much more marked than in others.

The accessory lesions, some of which are present in one case and some in others, are—

(1) A catarrhal inflammation of the mucous membrane of the bronchi.

(2) An exudative inflammation of the air-spaces, which fills their cavities with fibrin, pus, and epithelium, and produces consolidation of larger or smaller portions of the lungs. In young children the epithelial cells which line the air-spaces are much more numerous than they are in adults; so that when chil-

dren's lungs are inflamed the epithelial cells form a larger part of the inflammatory product than they do in the lungs of adults.

(3) An exudative inflammation of the pleura which coats it with fibrin.

(4) Dilatation of the bronchi, the walls of which are the seat of productive inflammation.

(5) Areas of atelectasis.

(6) Simple or tubercular inflammation of the bronchial glands.

As the inflammation of the walls of the bronchi and of the air-spaces surrounding them is from the first a productive inflammation, it follows the law which governs that form of inflammation. It is apt to last for a longer time than does an exudative inflammation, and it is liable to change into a chronic productive inflammation. It is not uncommon, therefore, for a broncho-pneumonia to continue for several weeks or to be followed by permanent changes in the lungs.

If the broncho-pneumonia becomes chronic, the inflammation of the walls of the bronchi and of the air-spaces which surround them continues; we then find that the bronchi are dilated, their walls are thickened; they are surrounded by zones of connective tissue, or part of a lobe or an entire lobe is changed into connective tissue. The pulmonary pleura may also be very much thickened.

Symptoms.—In very young infants the only symptoms are—fever, prostration, and rapid breathing. There is no cough; there are no physical signs. The disease is almost certainly fatal within a few days.

In older children the broncho-pneumonia may be preceded by the symptoms of measles, of whooping cough, of coryza, of pharyngitis, or of a catarrhal bronchitis of the larger tubes, or it may occur without having been preceded by any other morbid conditions.

There is a good deal of difference in the different cases as to the severity of the invasion. The more severe cases are ushered in by one or more general convulsions, or by a rapid rise of temperature, vomiting, oppressed breathing, and delirium. The milder cases begin with lower temperatures, moderate prostration, and increased frequency of breathing.

After the disease is established the patients continue to have a febrile movement. The temperature in most cases is irregular, but on many days up to 105° F. Very often the temperature is of distinctly remittent type—a morning temperature of 99° to 100° and an evening temperature of 104° to 105°. It is to be noticed, however, that in children both bronchitis and lobar pneumonia are also often accompanied by fever of a remittent type. It may very well be that this disposition to remission belongs rather to the age of the patient than to the character of the disease. The height of the temperature varies from day to day, sometimes with the progress or extension of the inflammation, sometimes without discoverable cause. In the cases which recover it requires several days for the temperature to fall to the normal. The height of the temperature is as a rule in proportion to the severity of the broncho-pneumonia: with temperatures of over 105° the mortality of the disease is considerable. There are, however, patients in whom the temperature runs between 99° and 100° who do very badly.

In children the pulse is normally more rapid than it is in adults, and is also more easily rendered rapid by disease. So in broncho-pneumonia pulses of from 140 to 170 to the minute are not uncommon, and in bad cases the pulse can hardly be counted.

An increase in the frequency of the breathing is almost constantly present —as much as 40 to the minute even in mild cases, up to 60, 70, or 80 in the bad cases. It is of importance to notice not only the frequency of the breathing, but also how much air enters into the lungs. The breathing may be made worse for a time by distention of the stomach. Sleeplessness, restlessness, and delirium are often present, and sometimes very troublesome. They seem to depend partly on the fever, partly on the interference with breathing, partly on the temperament of the child.

The face is flushed, the tongue is coated and sometimes dry; there may be vomiting or diarrhœa; sometimes there is pain over the chest.

Cough is often present, dependent on the catarrhal bronchitis rather than on the broncho-pneumonia. The sputa are swallowed rather than coughed up. They may collect in the stomach and be vomited up.

The urine may contain a little albumin and a few casts, the kidneys being the seat of acute degeneration.

The physical signs vary with the condition of the lung. If the inflammation be limited to the walls of the bronchi and to the air-spaces immediately surrounding them, there are no physical signs except diminished breathing. If a catarrhal bronchitis be present, there are coarse and subcrepitant râles. If there be a diffuse pneumonia with consolidation of a considerable portion of the lung, there are dulness on percussion, bronchial breathing, and bronchophony. If there be fibrin on the pleura, there are crepitant or subcrepitant râles. The signs of the consolidation and of the pleurisy are usually developed between the second and fifth days, but it is not uncommon for them to be delayed until a much later period.

The duration of broncho-pneumonia in children varies very much in different cases. Of the fatal cases, the larger number die within two weeks, but some prove fatal within two days and some are protracted for seven or eight weeks. In the cases which recover the constitutional symptoms continue for from one to three weeks in the majority of the patients, but it is by no means unusual for the active symptoms to continue for six or eight weeks and yet the patients make a perfectly good recovery. Resolution requires a longer time than in lobar pneumonia—from seven to fourteen days in most of the cases.

The Cerebral Cases.—In many of the cases of broncho-pneumonia there are cerebral symptoms—convulsions, restlessness, and delirium—but in some patients these symptoms are developed to such a degree, and are so out of proportion to the pulmonary symptoms, that the cases require a separate description.

The symptoms resemble those of an acute or a tubercular meningitis. They may progress acutely with high fever, prostration, one or more convulsions, alternating delirium, and stupor: or the course is more subacute, with loss of appetite, vomiting, moderate prostration, not very high fever, alternating stupor and delirium. These symptoms may continue for from two to ten days before there are any pulmonary symptoms. Then, as the pulmonary symptoms are developed, the cerebral symptoms subside.

Persistent Cases.—If, after the subsidence of the acute broncho-pneumonia, a chronic inflammation persists, the children begin to improve, but yet do not get well.

In some the cough and the physical signs continue, the appetite is poor, the children do not gain in flesh and strength, but yet they are not sick in bed—often not confined to the house. In others the same symptoms exist; there is also an irregular fever, and the patients are sick in bed.

Of these protracted cases, some recover entirely; some recover with permanent consolidation of a portion of the lungs; some die exhausted by the disease; some are left with a chronic interstitial pneumonia which lasts for many years.

In some cases of acute broncho-pneumonia the accompanying inflammation of the bronchial glands may be of a tubercular character, and this may serve at a later period as the focus of infection which causes an acute general tuberculosis.

In adolescence the clinical picture of broncho-pneumonia is the same as it is in children, but the cerebral symptoms are not developed to the same extent, and the patients are more likely to cough up blood.

In adults the disease presents itself to us under several different forms:

1. The patient has an ordinary attack of catarrhal bronchitis lasting for several days. Instead of getting well promptly, however, the patients continue to cough and to feel sick, and on examining the chest we find a circumscribed area where there is dulness on percussion and loud, high-pitched voice. This consolidation of the lung does not, however, last very long, and the patients make a good recovery.

2. The patients are suddenly attacked with a very severe and general broncho-pneumonia. There are chills, a rapid rise in temperature, headache, pains in the back and chest, vomiting, great prostration, a rapid pulse which soon becomes feeble, very bad respiration—rapid, labored, and insufficient; venous congestion of the skin and of the viscera; cough at first dry, then with profuse mucous and blood-stained sputa; sleeplessness, restlessness, and delirium, and albumin in the urine. There are coarse, subcrepitant, and crepitant râles over both lungs, and sibilant and sonorous breathing; the percussion-note is normal or exaggerated or dull. The disease lasts for from seven to fourteen days; it is very apt to prove fatal.

The most efficient remedies are the energetic use of wet or dry cups over the entire chest, the administration of calomel or the sulphate of magnesium in small and repeated doses, ipecac, the inhalation of oxygen, stimulants, and hypodermic injection of nitro-glycerin.

3. There is a form of broncho-pneumonia in adults which resembles lobar pneumonia. There is a general catarrhal bronchitis, with broncho-pneumonia and consolidation of one or more lobes. The symptoms and physical signs are like those of lobar pneumonia, but with some difference. The invasion of the disease is not as sudden, the pulse is more rapid, the cerebral symptoms are more constant, the expectoration is like that of bronchitis, the physical signs are more slowly developed, the duration of the disease is rather longer, and resolution is slower.

4. There is a form of broncho-pneumonia which resembles tubercular broncho-pneumonia. The invasion of the symptoms is gradual and the disease is protracted over a number of weeks. The patients have more or less cough and expectoration—at first mucous, later muco-purulent, but not containing tubercle bacilli. There is a moderate fever, with evening exacerbations and sweating at night. The physical signs are those of bronchitis and consolidation of circumscribed portions of the lung. The patients have no appetite and lose flesh and strength. Often the diagnosis depends upon the examination of the sputa. After a number of weeks, in some of the patients the inflammation subsides and a complete recovery is made, but in others it continues and proves fatal.

5. Persons suffering from emphysema sometimes develop a subacute broncho-pneumonia, which may prove fatal.

6. Broncho-pneumonia, especially of the lower lobes, is secondary to the infectious diseases, to injuries, to surgical operations, and to any conditions which are likely to cause congestion of the lungs and the inhalation of streptococci.

Treatment.—If there be much general catarrhal bronchitis, the use of hot poultices or hot fomentations over the chest, the internal administration of small doses of ipecac and aconite at short intervals, and sometimes the use of small doses of calomel, are indicated.

For the cerebral symptoms the bromides or phenacetin, in moderate doses, are to be preferred, but it is sometimes necessary to use opium.

For the broncho-pneumonia, if it be severe, aconitine and digitaline can be employed as in lobar pneumonia.

Opinions differ as to the propriety of the use of stimulants. It is my own opinion that in children under five years of age brandy and whiskey do harm instead of good.

If resolution be delayed or if the broncho-pneumonia persist, we employ iron, quinine, the mineral acids, oxygen, cod-liver oil, and, above all, change of air.

THE PNEUMONIA OF HEART DISEASE.

Definition.—A chronic productive inflammation of the lungs, caused by chronic congestion, and resulting in thickening of the walls of the air-spaces, filling of their cavities with epithelial cells, and a deposition of pigment.

SYNONYM.—Brown or pigment induration of the lungs.

Etiology.—Any long-continued mechanical obstruction to the escape of the blood from the lungs into the left cavities of the heart can produce this form of pneumonia. The most frequent and effectual obstruction is furnished by a stenosis of the mitral valve, but any valvular lesion of the heart, dilatation of the ventricles, or aneurism of the arch of aorta may act in the same way.

Morbid Anatomy.—The first effect of the obstruction to the circulation seems to be a change in the capillary vessels in the walls of the air-spaces. These vessels become dilated, tortuous, and have their walls thickened. Then there is a gradual thickening and pigmentation of the walls of the air-spaces; an increase in the size and number of the epithelial cells, until they partly or completely fill the cavities of the air-spaces; and escape of the red blood-cells into the air-spaces. Finally, when the inflammation has reached its full development, there is smooth, red hepatization of portions of both lungs, an hepatization due principally to the filling of the air-spaces with epithelial cells.

The lungs are diminished in size, sometimes covered with old adhesions, but seldom with fibrin. There may be more or less serum of dropsical character in the pleural cavities. The texture of the lungs is leathery and dense or that of a smooth hepatization. They are dry, of a salmon-pink color mottled with brown or black. There may be large or small areas where the air-spaces are filled with extravasated blood.

Symptoms.—The principal symptoms exhibited by the patients are those due to the lesions of the heart or aorta. The changes in the lungs do not give as marked symptoms as might be expected. The physical signs are obscure—more or less dulness and diminished breathing. The rational symptoms are dyspnœa, cough, mucous and bloody sputa, or even the continued expectoration of pure blood.

Treatment.—It is evident that this condition of the lungs is one which cannot be influenced by treatment. We simply attend as well as we can to the disturbances of circulation which have caused the lung trouble.

INTERSTITIAL PNEUMONIA.

Definition.—A chronic productive inflammation which involves the connective-tissue framework of the lung and the walls of the air-spaces, and results in the formation of new connective tissue and obliteration of the air-spaces.

It is customary to describe under the name of "interstitial pneumonia" the forms of productive pneumonia which follow acute lobar pneumonia with the formation of connective tissue, broncho-pneumonia, chronic pleurisy, chronic bronchitis, and the inhalation of the dust of coal or of stone. The forms of productive pneumonia which are associated with the growth of tubercle bacilli or of actinomyces, those which are caused by the poison of syphilis, and those which belong to pulmonary emphysema are usually described under other names.

Morbid Anatomy.—The condition of the lungs varies with the cause of the interstitial pneumonia:

(1) If it follow acute lobar pneumonia with the production of new connective tissue, one lobe or an entire lung is covered with pleuritic adhesions; it is small, smooth, and dense; the air-spaces and small bronchi are obliterated by the new connective tissue. Some of the bronchi may be dilated.

(2) If it be consequent upon broncho-pneumonia, one or more lobes are studded with fibrous nodules or are converted into dense fibrous tissue. The pleura is thickened, the bronchi are inflamed and often dilated.

(3) If it be concomitant of thickening of the pleura, bands of connective tissue extend from the pleura into the lung; the bronchi are inflamed and often dilated.

(4) If it follow chronic bronchitis, there are fibrous nodules around the bronchi, with more or less diffuse connective tissue.

(5) If it be due to the inhalation of the dust of coal or stone, we find in both lungs fibrous peribronchitic nodules and diffuse connective tissue.

In most of the cases the portions of lung exempt from the interstitial pneumonia are emphysematous.

Symptoms.—The patients have a cough with mucous expectoration. The cough becomes more constant and troublesome as the disease progresses. The expectoration becomes muco-purulent, sometimes fœtid. If the bronchi become dilated, the muco-pus accumulates in them and is coughed up at intervals in large quantities. There may be occasional hæmoptyses. There is dyspnœa on exertion, at first slight, later more marked. There may be uncomfortable feelings or actual pain over the affected side of the chest. There is gradual loss of flesh and of strength. Neither laryngitis nor diarrhœa belongs to the disease. There is no fever except with exacerbations of the bronchitis.

In the cases of interstitial pneumonia which follow lobar pneumonia, broncho-pneumonia, and chronic pleurisy the affected side of the chest is retracted, the other side is enlarged, the heart is displaced, the vertebral column is curved laterally. There is, on percussion, dulness or flatness or tympanitic resonance. The vocal fremitus is increased. The breathing is sonorous, sibilant, bronchial, cavernous, or amphoric according to the condition of the bronchi and the degree of consolidation of the lung. There are pleuritic creaking sounds, subcrepitant, coarse, or gurgling râles.

The disease is one which lasts for many years, and the patients usually die with some acute inflammation of the other lung.

Treatment.—The most efficient treatment is that the patient should reside permanently in a climate where he is able to live out of doors and where his bronchitis does well. If this cannot be done, we attend to the bronchitis and the nutrition of the patient as well as we can.

TUBERCULAR PNEUMONIA.

Definition.—Under this name we include all the inflammations of the lung which are accompanied with the presence of tubercle bacilli. Of such inflammations there are a number which differ from each other widely in their morbid anatomy and clinical symptoms. We are not at the present time provided with satisfactory names to designate these different forms of tubercular pneumonia, so that we still have to use the old arbitrary terms to which there are so many objections.

We distinguish, therefore, Acute Miliary Tuberculosis of the Lungs; Chronic Miliary Tuberculosis of the Lungs; Acute Pulmonary Phthisis; Chronic Pulmonary Phthisis.

ACUTE MILIARY TUBERCULOSIS OF THE LUNGS.

Definition.—A tubercular inflammation of the lungs characterized by the presence of a number of small foci of inflammation, of which the inflammatory products form very small nodules called miliary tubercles. The tubercular inflammation may be confined to the lungs or it may be part of a general tuberculosis.

(a) *Secondary Form.*

Etiology.—For the development of tubercular inflammation in any part of the body there are necessary the proper predisposition of the individual, a local cause of inflammation, and the growth of tubercle bacilli. In the lungs the tubercle bacilli seem to be either inhaled or transported by the blood. It is possible that the bacilli may be first inhaled, then collected in the bronchial glands, and from the glands find their way into the pulmonary circulation. Certainly, we see cases in which tubercular inflammation of the bronchial glands precedes miliary tuberculosis of the lungs. The bacilli which are inhaled must be derived to some extent from the dried sputa of persons who have tubercular pneumonia. The bacilli are much more abundant in the air of some localities than in that of others, and at high altitudes and on the ocean the air contains none of these organisms. The bacilli which are conveyed to the lungs by the blood are derived from a focus of tubercular inflammation in some other part of the body. In man tuberculosis is conveyed by inoculation from one person to another only in rare cases. In some animals tuberculosis can be inoculated at will, and miliary tubercles of the lung easily produced. Trudeau, however, has shown that the success of such an inoculation can be influenced by environment, and that it is possible to keep rabbits under such conditions of good health that they cease to offer a good soil for the growth of tubercle bacilli. The most perfect miliary tubercles in the lungs of animals which I have ever seen are those produced by injections of dead tubercle bacilli into the trachea of rabbits.

Morbid Anatomy.—The miliary tubercles may be confined to part of a lung or be distributed through both lungs. They are close together, or separated by considerable intervals, or aggregated together into larger masses. They are of gray, white, or yellow color. They all contain tubercle bacilli, but these bodies are much more numerous in some tubercles than they are in others. They are composed of—

1. Groups of air-passages and air-vesicles filled with granular matter, pus-cells, and epithelium.

2. Groups of air-passages and air-vesicles of which the walls are infiltrated with tubercle tissue, while their cavities are filled either with tubercular tissue or with epithelium, pus, and fibrin.

3. Infiltrations of the walls of small bronchi with tubercular tissue or round-celled tissue, the infiltration extending to the walls of the adjacent air-spaces.

4. Nodules of tubercular tissue situated in the pulmonary pleura, the septa between the lobules, the walls of the bronchi, and the walls of the veins.

In addition to the presence of the miliary tubercles, the bronchi are congested and coated with mucus, the walls of the air-spaces are congested, the epithelial cells which line them are increased in size and number, some of the air-spaces are filled with epithelium, fibrin, and pus.

The most frequent position of the tubercles is at the apex of one lung, but they may be localized at any part of the lungs. It is not often that the whole of both lungs is involved. The cases vary greatly as to the extent of the associated bronchitis, pleurisy, and exudative pneumonia. The bronchitis may be confined to the portion of the lung which contains the miliary tubercles, or it may be a general bronchitis, involving the larger tubes in both lungs. The pleurisy is either with fibrin alone or with large quantities of serum in the pleural cavity. The exudative pneumonia may involve only small portions of the lung or an entire lobe may be consolidated.

Symptoms.—(1) The method of infection may be such that an enormous number of miliary tubercles are at once formed in the lungs and in other parts of the body; the poisonous effects of the chemical substances produced by the growth of the tubercle bacilli are very marked, and the patients behave as if they had a general disease rather than an inflammation of the lungs.

Although the lungs are congested and thickly studded with miliary tubercles, the physical signs are not as constant or as plain as one would wish them to be. The changes in the percussion note are not well marked. There may or may not be some dulness. Crepitant or subcrepitant or coarse râles, bronchophony, bronchial breathing, or rude breathing are sometimes present, sometimes absent. Moreover, the physical signs may be modified by the existence of old tuberclar lesions in the lungs. Rational pulmonary symptoms also are not constant. Cough and mucous sputa, rapid and oppressed breathing, and pain over the chest are present in some cases, absent in others.

The temperature rises rapidly and is between 100° and 103° F. throughout the disease. The pulse and heart-action become more rapid and feeble as the disease progresses. The tongue is coated, and soon becomes brown and dry. There is loss of appetite, nausea, and sometimes vomiting. The bowels remain regular or are constipated or loose. Sooner or later alternating stupor and delirium, extreme emaciation, and the typhoid state are developed. The disease may last for only a few days or for three or four weeks. So far as I know, it always terminates fatally.

(2) The method of infection is such that miliary tubercles are formed only in part of a lobe, or are distributed through an entire lobe or the whole of one lung or a large part of both lungs. But there are no tubercles outside of the lungs; it is a localized tubercular inflammation.

This condition may be developed at any time in a person who has the tubercular disposition, and we are seldom certain in such cases as to the exciting cause of the inflammation or the exact method of the infection. It is also of frequent occurrence in persons who already have a chronic tubercular inflammation of some part of the lung. In such cases it is probable that the infection comes from the old tubercular lesion.

The symptoms are in proportion to the extent of lung involved. When the disease is established the patients suffer from:

A febrile movement which is regularly higher in the afternoon and followed by sweating at night. The fever may be confined to the afternoon, with normal morning temperatures. The height of the temperature varies with the extent of lung involved, the severity of the associated bronchitis, pleurisy, or pneumonia, the character of the patient, and, probably, the quantity of poisonous products which are evolved by the growth of the bacteria.

An increased frequency of the heart-action. This is a very constant symptom, so constant that whenever we find a patient with a rapid pulse for which there is no evident cause, we must always think of the possibility of a tubercular inflammation of the lungs.

Respiration is often increased in frequency, and sometimes labored, even with lesions of small extent. It is made worse by an extension of the tuberculosis or by the development of bronchitis, pleurisy, or pneumonia.

Most of the patients have more or less cough. They may cough only in the morning, or throughout the day, or in severe attacks which may provoke vomiting. In some patients the cough is hardly noticeable; in others, it is the most distressing feature of the disease. The immediate cause of the cough is not always the same. It may be principally due to a catarrhal pharyngitis or to the bronchitis or to the pleurisy. How far the presence of the tubercles in the lungs causes cough is difficult to determine. In some persons the cough is evidently largely hysterical, out of all proportion to any real reason for it. The expectoration, if there be any, is mucous or muco-purulent in character. It often, but not always, contains tubercle bacilli. There may be hæmoptyses throughout the course of the disease, or they may precede all the other symptoms.

Loss of appetite with more or less nausea is present in a great many of the patients. It is a serious matter, for it is one reason for their loss of flesh and strength. Actual vomiting usually occurs only after fits of coughing.

Loss of flesh and strength are characteristic symptoms, but they are not always in proportion to the extent of lung involved. We must judge of the real condition of the patient rather by the pulmonary lesions than by the general condition.

It is questionable whether the miliary tubercles alone give any physical signs; and as a matter of fact, many patients who have acute miliary tuberculosis give no physical signs at all. The associated bronchitis, pleurisy, and pneumonia do, however, give physical signs. The bronchitis gives coarse and subcrepitant râles; the pleurisy, dulness, friction-sounds, crepitant and subcrepitant râles; the pneumonia, dulness, increased vocal resonance, bronchophony and bronchial breathing.

Course of the Disease.—(1) When the disease is once established it may continue for a number of months, the inflammation then subside, and the patients recover altogether.

(2) Or the inflammation may remain localized, but will become chronic and the patients suffer from of chronic miliary tuberculosis.

(3) Instead of this, the tubercular inflammation, after remaining for months circumscribed, will either continuously or at intervals extend and involve more and more of the lungs. When this is the case the patients grow worse either continuously or with intervals of improvement.

(4) In some cases there are one or several intercurrent attacks of bronchitis, pleurisy, or pneumonia so severe as to modify the course of the disease. With each attack of this kind the temperature rises, the physical signs change, and the patient is evidently more seriously ill.

(5) If both lungs are at the first involved by the tubercular inflammation, the patients have the same symptoms, but in a more severe form. They lose flesh and strength rapidly, develop alternating delirium and stupor, pass into the typhoid condition, and die within a few weeks or months.

Treatment.—If both lungs are involved in the morbid process, treatment is of no avail. If, however, only a part of one lung is diseased, the patients can be much benefited by proper management, the two essential points in the treatment being that the patients should eat enough food and live in a proper climate.

As regards the feeding, it is important that the patients should take fat in some form in addition to the other articles of diet. In some patients a proper climate alone will improve the appetite; in others it may be necessary to use bitters, alkalies, or mineral acids, to relieve constipation, to wash out the stomach, or to feed through the stomach-tube. Generally speaking, all the prepared and peptonized foods and extracts are to be avoided. The patients do best with milk, cream, meats, vegetables, fruits, and breadstuffs.

As regards climate, I doubt if there is any one place suitable for all the patients. The idiosyncrasy of each person must be considered, and we find that some do best on the sea-shore, some in the interior, some in a warm climate, some in a cold climate, while some do best if they travel from place to place.

Of the different drugs and inhalations used for the direct treatment of the tubercular inflammation, none seems to be capable of killing the tubercle bacilli or of acting as an antidote to the poison produced by them, although great expectations have been entertained concerning such remedies.

The empirical use of beechwood creasote, or of its equivalent guiacol, or of the carbonate of guiacol, still continues to give some fairly good results. Physicians differ as to their preference for one or other of these preparations, as to the size of the dose, and as to the method of administration. For my own part, I prefer creasote given by inhalation. A small perforated zinc nosepiece (Robinson's), of which the sponge is kept moistened with a mixture of equal parts of creasote, alcohol, and chloroform, can be worn nearly all the time. If the creasote be given by the mouth, 5 minims is a sufficient dose, and cod-liver oil is the best vehicle.

Of the symptoms, the cough is the one which most frequently calls for treatment. If the cough be caused by pharyngitis or laryngitis, local treatment is to be preferred. If due to pleurisy, counter-irritation is of service. If dependent on bronchitis, we employ the usual remedies for this inflammation.

CHRONIC MILIARY TUBERCULOSIS.

Definition.—A chronic tubercular inflammation of the lungs characterized by the formation of miliary tubercles, to which may be added bronchitis, dilatation of the bronchi, pleurisy, interstitial pneumonia, and emphysema.

Etiology.—It seems to be necessary to suppose in these cases a predisposition, an exciting cause, for inflammation and the growth of tubercle bacilli. It is evident, however, that the mode and character of the infection must differ from that of acute miliary tuberculosis. The structure of the tubercles is different; they contain very few tubercle bacilli, and the other morbid changes in the lungs may be of more importance than are the tubercles.

Morbid Anatomy.—The morbid process begins as a rule at the apex of one of the lungs, and then slowly extends either progressively or in attacks until a large portion of the lungs is involved.

In the simplest form of the disease the only change in the lungs is the formation of miliary tubercles. These tubercles are harder and denser than are those which are found with acute tuberculosis. They are composed of tubercle tissue or round-celled tissue or connective tissue, or are in the condition of cheesy degeneration. They contain but few tubercle bacilli.

Another simple form of the disease is when miliary tubercles alone are formed in lungs which are already the seat of vesicular emphysema.

Usually, however, in addition to the miliary tubercles there are other changes in the lungs. These additional lesions begin in the same part of the lung where the tubercles are formed, and accompany the development of the tubercles in fresh parts of the lungs.

(1) There may be a localized catarrhal bronchitis.

(2) There may be an inflammation of the walls of the bronchi with partial destruction of these walls and the formation of cylindrical or sacculated bronchiectasiæ. The walls of the cavities thus formed may be converted into connective tissue or they may remain suppurating and necrotic.

(3) There may be an interstitial pneumonia, with the production of new connective tissue, the obliteration of the air-spaces, and the consolidation of portions of the lung.

(4) There may be dilatation of the air-spaces of the portions of the lungs which are not consolidated.

(5) There may be thickening of the pulmonary and costal pleura, with connective-tissue adhesions.

(6) While the morbid process begins as a localized tubercular inflammation of the lung, and often retains throughout this local character, yet it may also happen that from this local lesion other parts of the body may be infected. Tubercular laryngitis and tubercular inflammation of the solitary and agminated glands of the small intestine often complicate the pulmonary lesion, and sometimes even acute general tuberculosis is produced.

Symptoms.—The disease may follow an acute pulmonary tuberculosis or it may be chronic from the outset. There is such a very great difference in the behavior of the disease in different persons that it is necessary to arrange the cases into a number of groups according to the character of the lesions and the symptoms:

1. There seems good reason to believe that a small tubercular inflammation of one apex often runs its course and terminates in recovery without ever giving symptoms of sufficient severity to attract the attention of the patient or lead him to consult a physician.

2. There are patients who suffer for some time from pulmonary emphysema with its attendant symptoms. Then miliary tubercles are formed in the lungs and slowly increase in number, but are not accompanied with the growth of much connective tissue, and are scattered at some distance from each other through the lungs. The formation of the tubercles does not change the physical signs belonging to the emphysema which the patient already has. The ordinary rational symptoms of emphysema continue unchanged, but the patients lose flesh and strength rapidly, and finally die very much emaciated. It is very difficult to distinguish these cases from the bad cases of emphysema without tuberculosis, although the examination of the sputum may help us by revealing the tubercle bacilli.

3. A very common form of the disease is that in which the inflammation is confined to one or both apices, continues for some time, subsides, and the patients recover.

In these cases the first symptom may be one or more small or large hæmoptyses. Before the bleeding the patients have had no pulmonary symptoms, but after the bleeding these symptoms are gradually developed. Or the first symptom may be a troublesome cough with little or no expectoration. This cough at first does not seem of much consequence, but it continues in spite of all remedies. Or the patients simply lose flesh and strength without any evident reason for this loss of nutrition. The pulse is also increased in frequency, but for a time there are no pulmonary symptoms.

At this early stage of the disease there may be no physical signs. As the disease advances the patients have more or less cough, which troubles them only in the morning or occurs in attacks, or is persistent and troublesome through the whole day. There may be no expectoration. When present the sputa are mucous or muco-purulent, not abundant, and often contain tubercle bacilli. There is some dyspnœa on exertion. Some of the patients complain of a great deal of pain over the inflamed lung, others have no pain at all. Hæmoptyses may be repeated at any time, and are apt to be followed by an increase in the severity of the symptoms. The frequency of the pulse is increased in nearly all the patients. A febrile movement is not a prominent symptom; there may be afternoon temperatures of 100° F., followed by sweating, but often during much of the time there is no fever at all. The appetite is poor, nausea and vomiting are often troublesome. The patients lose flesh and strength. In some cases the symptoms are not at any time severe; in others the constitutional disturbances are so out of proportion to the lesion as to indicate systemic infection.

The physical signs become more marked with the further development of the thickening of the pleura, the formation of miliary tubercles, the growth of interstitial connective tissue, the localized bronchitis, and the dilatation of the bronchi. We find retraction of the chest-wall above and below the clavicle, the percussion sound of higher pitch and of shorter duration above and below the clavicle, subcrepitant râles, friction sounds, a high-pitched and louder transmission of the voice, the breath-sounds diminished or of altered quality or with prolonged expiration.

Such a circumscribed tubercular inflammation usually continues for a year, sometimes longer; then it subsides and the patients recover. The portion of lung which has been inflamed is left permanently changed into connective tissue.

As the patient has had one attack of tubercular inflammation, so, although he has entirely recovered from this attack, he may have subsequent attacks of the same kind. In some of the patients after the subsidence of the inflammation tubercle bacilli are left in the portion of lung which has been inflamed. These may at any time later serve as a source of infection for a new local or general tuberculosis.

4. The tubercular inflammation, beginning at the apex of one lung, gradually extends and involves a large part of both lungs. The pleuritic adhesions become more extensive, a larger number of bronchi are involved in the catarrhal bronchitis, miliary tubercles and new connective tissue replace more and more of the lung tissues. The changes in the walls of the bronchi and of the lung surrounding them result in the formation of bronchiectatic cavities, which constantly increase in size and the walls of which are necrotic or suppurating. As the disease progresses, therefore, the patients suffer not only from the infection due to the tubercular inflammation, but also from that due to the necrosis and suppuration of the walls of cavities, while in addition more and more of the lungs is rendered unfit for breathing.

The physical signs of consolidation and of pleuritic adhesions become more marked, and as the cavities are formed and increase in size their physical signs are added.

The cough, which depends at first upon the bronchitis or the pleurisy, is made worse by the formation of bronchiectatic cavities. The expectoration becomes more profuse, more purulent, and contains portions of necrosed lung. Bacilli are present in the sputum in larger numbers. The difficulty in breathing becomes more troublesome. The patients differ very much as to the presence or absence of pain in the chest. Either large or small hæmoptyses may be repeated at any time. At first the blood comes from the mucous membrane of the bronchi, but after cavities have been formed there may be bleeding from eroded vessels in their walls. This bleeding is apt to be profuse, continuous, and often fatal. The pulse continues to be rapid through the greater part of the disease. The fever becomes higher and more continuous as the disease progresses, especially after the formation of cavities. The appetite is poor, and nausea and vomiting are often troublesome. In women menstruation becomes irregular or ceases altogether. The patients get worse from year to year, but often with periods of improvement, and the whole duration of the disease is apt to be very considerable.

After a time, in many of the patients a tubercular inflammation of the larynx or of the agminated glands of the small intestines is added, and then the loss of flesh and strength are much more rapid.

5. There are cases in which the tubercular inflammation gradually extends until both lungs are thickly studded with miliary tubercles, and in addition there are extensive pleuritic adhesions, but there is little diffuse fibrous tissue, little or no bronchitis, and no dilatation of the bronchi.

The clinical history of these patients is very misleading. They have no cough, no hæmoptyses, no pulmonary symptoms, no fever. There are no physical signs except those belonging to the pleuritic adhesions. But very often the functions of the stomach and intestines are much disordered. The most striking symptom of these patients is their emaciation. This progresses steadily until the patients are mere skeletons, looking as if they were starving to death with a cancer of the stomach; and yet they may really be taking and retaining a considerable quantity of food. In spite, however, of the food, they continue to lose flesh as if they had a malignant disease. The diagnosis of these cases is often extremely difficult.

6. There are cases in which a tubercular laryngitis is responsible for most of the symptoms, the lesions in the lungs being inconsiderable. The upper part of the larynx and the epiglottis are the portions usually involved. There is first a formation of tubercle-granula here and there in the stroma of the mucous membrane, with more or less catarrhal inflammation. After a time the tubercle-granula and the mucous membrane over them become necrotic, soften, slough, and form ulcers. These ulcers do not heal, but rather increase in size, their floors and walls being formed partly of tubercle, partly of round-celled tissue. The mucous membrane left between the ulcers is thickened and the seat of chronic catarrhal inflammation.

With such a tubercular laryngitis the patients have a cough with mucopurulent expectoration, a changed voice, and sometimes a laryngeal dyspnœa. But the thing which troubles them the most is the pain in the throat, which is made worse by swallowing. This may interfere seriously with the feeding of the patient.

The patients lose flesh and strength, but are not confined to bed. Indeed, many years may pass before the pulmonary tuberculosis gives much trouble.

7. In a few cases a very small miliary tuberculosis of the lung is complicated with an extensive tubercular inflammation of the small intestine.

These patients have a little cough, and perhaps the physical signs of a small consolidation of one apex. It is important to remember that they do not necessarily have diarrhœa. But they lose flesh and strength with a rapidity which the pulmonary conditions do not account for.

Prognosis.—In the early periods of the disease with little evidence of systemic infection we may often expect the complete recovery of the patients. As more of the lungs is involved, as cavities are formed, as the complicating laryngitis and enteritis are developed, and the evidences of systemic infection become manifest, the prognosis is worse and worse.

Treatment.—The curative treatment of chronic miliary tuberculosis is embraced in two principal things—climate and feeding. The selection of a proper climate is to be made with reference to the individual rather than to the condition of the lungs. It should be a climate where he feels well, eats well, sleeps well, and gains flesh and strength. If no one climate answers this purpose, the patient should travel from place to place. The climatic treatment should be continued, if possible, for two full years, and for some persons it is necessary that they should pass the rest of their lives in a favorable climate.

The feeding consists in enabling the patients to eat and digest considerable quantities of wholesome food and of fats. To do this it is necessary to pay the most minute attention to the functions of the stomach, the liver, and the intestines. Great care should be taken to avoid the use of all medicines which interfere with the patient's ability to eat and digest food. Wines and spirits will with some persons increase the appetite and the nutrition; with other persons they interfere with digestion and do harm. Often we are much helped by the use of the stomach-tube. Not only can we in this way cure a complicating chronic gastritis, but we can introduce into the stomach much larger quantities of fluid food than the patients are willing to swallow.

It may also be necessary to alleviate symptoms. The cough is not only annoying, but it often interferes with eating and sleeping. It is therefore important in each case to determine the principal cause of the cough. It may be due to a catarrhal inflammation of the nose and throat, or to either a catarrhal or tubercular inflammation of the larynx. These conditions are best treated by local applications made with the spray or with the brush. It may be due to the pleuritic adhesions. If this be the case, counter-irritation by blisters or iodine may be of service, but some of these pleuritic coughs can only be controlled by opium.

For the cough which is due to the bronchitis and to the cavities a great many remedies have been employed. In selecting from these remedies it is well to prefer those which do not disorder the stomach. Creasote seems to be capable of exerting a real effect on the bronchitis, but, unfortunately, it is apt to disorder the stomach. I prefer to use it by inhalation or by enema. For inhalation a mixture is made of equal parts of creasote, chloroform, and alcohol. The sponge of a Robinson's inhaler is moistened with a few drops of this mixture, and the patient wears the inhaler all the time except when sleeping or eating. Many patients object, however, to the long-continued use of the inhaler each day. An enema is easily made up of 5 to 20 drops of creasote with some white of egg and a little water, and this can be used once a day. The different preparations of tar and of turpentine, terebene and terpin hydrate, seem to be of service in some cases. The methodical inhalation of compressed air is highly thought of by some physicians. All sorts of combinations of opium, ipecac, squills, sanguinaria, hydrocyanic acid, chloroform, senega, etc. are given as cough mixtures. The mineral acids, nux vomica or strychnine, and potassium iodide may somewhat control the bronchitis.

If the patients are anæmic, they may be benefited by one of the preparations of iron.

The hæmorrhages from the bronchi may be small or large, but even if large and continued for several days, they are very seldom fatal. They do, however, weaken the patient very much, and are often followed by an extension of the tubercular pneumonia. To check such hæmorrhages it is customary to use hypodermic injections of morphine or of ergotine, or to give by the mouth 5 grains of gallic acid every two hours, 20 drops of fluid extract of hydrastis every three hours, or 1 grain of ipecac every hour.

The hæmorrhages which come from eroded vessels in the walls of cavities cannot be controlled.

The fever and night-sweats may be made less severe by the use of antifebrin or phenacetin alone or combined with arsenic or quinine; by the mineral acids, belladonna, or the oxide of zinc; by sponging the body with hot water at night.

For the diarrhœa we employ a restricted diet and a number of drugs. Those most frequently employed are the preparations of mercury, ipecac, iron, arsenic, camphor, acetate of lead, bismuth, castor oil, opium, and naphthalin. These drugs are used singly or combined in different ways.

ACUTE TUBERCULAR PHTHISIS.

SYNONYMS.—Acute catarrhal phthisis; Acute consumption.

Definition.—An acute affection of the lungs characterized by the association of tubercular inflammation with other forms of inflammation, either exudative, productive, or both. The name is an arbitrary one, and is used for convenience to group together a set of clinical cases.

Etiology.—An attack of acute phthisis may follow some previous tubercular inflammation of the lung or it may be a primary inflammation. A person who has the tubercular predisposition when exposed to the ordinary causes of inflammation of the lung, and at the same time infected with tubercle bacilli, instead of having a simple exudative or productive pneumonia, has inflammation of the lung, partly tubercular, partly exudative, partly productive.

Morbid Anatomy.—The inflammation of the lungs may follow one of several types, all of which have much the same clinical history, but vary in their physical signs.

1. One or more lobes are completely consolidated. The consolidation is effected by the filling of the air-spaces and small bronchi with epithelium, fibrin, and pus. Scattered through the consolidation are miliary tubercles. The pleura is coated with fibrin.

2. There is a general catarrhal bronchitis and a tubercular inflammation of the walls of some of the bronchi and of small zones of air-spaces immediately surrounding them. The lung is not consolidated, but a section of it appears to be studded with little nodules; each nodule is the section of a bronchus with thickened wall and surrounded by a zone of filled air-spaces. It is a tubercular broncho-pneumonia.

3. There is a general catarrhal bronchitis, a tubercular inflammation of the walls of some of the bronchi and of the air-spaces which surround them; but in addition there are small or large areas of diffuse consolidation, due to the filling of air-spaces with epithelium, pus, and fibrin. The pulmonary pleura is often coated with fibrin. It is a tubercular broncho-pneumonia with the addition of diffuse consolidation.

4. Besides the tubercular broncho-pneumonia, the diffuse consolidation, and the pleurisy, small or large portions of the inflamed lung die. These dead portions of lung first pass into the condition of coagulation necrosis, and then undergo cheesy degeneration. They may remain in the condition of cheesy degeneration for a long time, and become surrounded by zones of tubercle tissue or of round-celled tissue, or they soften, break down, and form ragged cavities which communicate with the bronchi.

5. In addition to the lesions already mentioned, the walls of the bronchi are so changed by the tubercular inflammation that cylindrical or sacculated bronchiectatic cavities are formed.

Symptoms.—The invasion may be acute or subacute.

1. *The Acute Cases.*—The patients are suddenly attacked with chills, fever, pain in the side, cough with mucous expectoration, and marked prostration. The appearance of the patient is like that of a person attacked with lobar or broncho-pneumonia, and we are often in doubt at first as to the true nature of the disease. One symptom of the invasion, however, is not often seen except with phthisis, and that is the bleeding from the bronchi. For a day or for several days many of the patients cough up very considerable quantities of blood. Within a few days we begin to get the physical signs, which will be found to vary according to the anatomical condition of the lung. If there be complete consolidation of one or more lobes with fibrin on the pleura, we get dulness on percussion, bronchial breathing and bronchophony, and subcrepi-

tant or crepitant râles. If there be only broncho-pneumonia without consolidation, we get sibilant and sonorous breathing and coarse and subcrepitant râles. If there be broncho-pneumonia with areas of diffuse consolidation, we get sibilant and sonorous breathing or harsh breathing, coarse and subcrepitant râles, and small areas over which there are dulness on percussion, increased voice, and a crepitant râle. The patients continue seriously ill and with high temperatures for one or two weeks. Then there is a partial subsidence of the symptoms. After this the cases may pursue various courses—

(*a*) They may continue to get worse. The fever continues; the cough is very troublesome and accompanied with muco-purulent expectoration; there is rapid loss of flesh and strength; the patients pass into the typhoid state with alternating delirium and stupor. The physical signs which existed earlier in the disease continue, and there are added the coarse and gurgling râles which accompany the dilatation of the bronchi and the softening of the dead areas of consolidation. These patients die at the end of a few weeks or months.

(*b*) They may improve very considerably. The temperature falls, the expectoration diminishes, the cough is less frequent, the appetite returns. The patients gain flesh and strength; they are able to leave the bed, and later the house, but yet they are not well. A considerable portion of the lung remains diseased, and the patients lapse into the condition of chronic phthisis.

(*c*) Recovery may occur. Of the lesions of acute phthisis, the tubercular changes, the death of portions of lung, and the dilatation of the bronchi are necessarily permanent. On the other hand, the catarrhal bronchitis may subside; the epithelium, pus, and fibrin within the air-spaces may degenerate and be absorbed. It is possible, therefore, for the patients who have only catarrhal bronchitis, exudative pneumonia, and comparatively little tubercular pneumonia, to recover. We see this in two sets of cases:

First: The patients who have consolidation of an entire lobe due to the filling up of the air-spaces with epithelium, pus, and fibrin and to the presence of miliary tubercles. The epithelium, pus, and fibrin can be absorbed, the tubercles are converted into fibrous tissue, and the patients get well with a lung which is normal except for the presence of fibrous nodules.

Second: The patients who have tubercular broncho-pneumonia without destruction or dilatation of the bronchi and without areas of diffuse consolidation. These patients can get well with lungs which are normal, except for the presence of a number of peribronchital fibrous nodules.

2. *The Subacute Cases.*—The extent of lung at first involved is small, but gradually increases in size. The patients usually have cough with mucous or muco-purulent sputa, which contain bacilli; but sometimes there is very little either of cough or of expectoration, and that even while cavities are being formed. The difficulty in breathing increases with the extent of lung involved. Pain over the chest is present in some cases, absent in others. There is as a rule a rise of temperature in the afternoon, with sweating at night, and the temperature is higher and more continuous after cavities with

suppurating walls are formed. We have to become accustomed, however, to very great discrepancy between the lung lesion and the height of the temperature, and we may even find no fever with an advancing consolidation of the lung.

There may be bleeding from the bronchi or from eroded vessels in the walls of cavities. A large hæmorrhage from the bronchi is apt to precede the inflammation of a fresh portion of the lung. The patients, as a rule, have no appetite, and gradually lose flesh and strength ; but in some persons the changes in the lung for some time produce very little effect on the general health. It is sometimes very curious, and of importance in judging of treatment, to see patients eating well and gaining flesh with consolidation of an entire lung, and without any real improvement in their tubercular pneumonia.

The physical signs are those of the pleurisy, the consolidation, the bronchitis, and the cavities. The patients are, for the most part, not sick in bed, and the disease progresses either continuously or by exacerbations.

Some of the patients become the subjects of chronic phthisis. Some of them recover, but with lungs more or less permantly damaged : often these patients can only escape fresh attacks by remaining permanently in a favorable climate.

The **prognosis** of acute phthisis is unfavorable. Complete recoveries are rare, but the number of patients who continue to live with damaged lungs for many years is considerable.

Treatment.—While the inflammation of the lungs is active the patients are to be kept in bed on a fluid diet, the hæmoptyses controlled by ergot, hydrastis, or ipecac, and the patients made more comfortable by opium or the bromides. As the acuteness of the inflammation subsides the patients may return to solid food, and get out of bed, and then the question of a suitable climate for them has to be determined.

In some cases it is evident that the changes in the lungs are so extensive and profound that no improvement can be expected ; these patients are best kept at home.

In some cases the extent of lung involved is comparatively small, so that we may hope for complete or incomplete recovery. The proper climate for these patients is a dry, inland one, where they are not likely to have fresh attacks of inflammation of the lung. Whether this climate should be a warm or cold one must be determined by the character of the individual. In the cases of tubercular broncho-pneumonia without consolidation a cold, dry inland climate seems to be the best.

The symptomatic treatment is the same as that used in the cases of acute miliary tuberculosis.

CHRONIC TUBERCULAR PHTHISIS.

Definition.—A chronic affection of the lungs characterized by tubercular inflammation, associated with productive and exudative inflammation.

Etiology.—Chronic phthisis as a rule succeeds acute or subacute phthisis, but it may also follow acute or chronic miliary tuberculosis of the lungs or a tubercular inflammation of some other part of the body

Morbid Anatomy.—The changes in the lungs are of the same nature as those found in acute phthisis, but modified by the long duration of the inflammation. The pleura is coated with successive layers of fibrin, or thickened by the growth of new connective tissue, or covered with adhesions. More or less of the lung is consolidated. This consolidation is effected partly by the filling of the cavities of the air-spaces with inflammatory products, partly by a growth of new tissue in and between the walls of the air-spaces.

When the hepatization is effected only by changes within the cavities of the air-spaces, the affected portions of lung are solid, increased in size, of red, gray, white, or yellow color. When the hepatization is due to interstitial inflammation, the affected portion of lung is dense, but may still be partly permeable by air; it is diminished in size and looks like fibrous tissue or granulation tissue; it is often changed in color by the deposition of black pigment. The combination of intra-alveolar and interstitial pneumonia with dilatation of the bronchi and the formation of cavities gives a great variety of pictures. We find some of the air-spaces filled with large epithelial cells, either well formed or fatty; some with an amorphous granular matter or a peculiar, translucent coagulated substance; some with fibrin, pus, and epithelium, either fresh or in the condition of cheesy degeneration; some with new connective tissue. The walls of these air-spaces remain unchanged, or they are compressed and the blood-vessels obliterated, or they are thickened.

The interstitial inflammation affects the walls of the air-spaces, the bronchi, the blood-vessels, and the septa between the lobules. It results in the production of new connective tissue, of round-celled tissue, and of tuberculous tissue, either separately or together. By this growth the air-spaces are compressed, deformed, and obliterated in a variety of ways.

The walls of some of the bronchi are infiltrated with round cells or with tuberculous tissue. This infiltration is not symmetrical, but affects a bronchus in some particular portion of its length, and in this portion some parts of the circumference of the bronchus are affected more than others. As a result of this irregular infiltration the wall of the bronchus yields here and there, and small sacculated dilatations are formed. In some cases, especially in chronic miliary tuberculosis, the cavities thus formed simply become larger and larger, compressing the surrounding lung. More frequently, however, the process extends from the wall of the bronchus to the surrounding air-vesicles, so that the bronchus is surrounded with tuberculous tissue, round-celled tissue, and air-vesicles filled with inflammatory products. Then necrosis sets in with softening of the walls of the bronchus and of the surrounding inflamed lung. Cavities are thus formed partly by destruction of tissue, partly by dilatation of bronchi of which the walls are either necrotic or suppurating.

Some of the cavities in chronic phthisis seem to be formed simply by the softening of areas of coagulation necrosis, but the larger number are bronchiectatic.

The cavities which are formed by dilatation without necrosis of their walls may be developed with hardly any cough or expectoration. As we examine the patients from time to time, the change in the percussion-note and the breath-sounds shows the increasing size of a cavity which remains nearly dry and empty.

The cavities formed both by dilatation and necrosis, on the contrary, are as a rule accompanied by a harassing cough and profuse expectoration. They contain pus, mucus, fragments of dead tissue, and great numbers of tubercle bacilli. Their walls are ragged and irregular, partly necrotic, partly suppurating. In this active condition the cavities may remain up to the time of the patient's death; or, instead of this, the active processes may subside, the production of pus and the death of tissue cease, the cavity become dry, and its walls changed into fibrous tissue. The natural tendency of all these cavities is to increase in size and open into each other.

Miliary tubercles are scattered through the inflamed lung in varying numbers.

The tubercle bacilli are found principally in the walls of the cavities and in the inflammatory products which have undergone cheesy degeneration.

Symptoms.—The cough depends principally upon the bronchitis and the morbid processes going on in the walls of cavities. The expectoration consists of mucus, pus, and fragments of dead lung-tissue, with many tubercle bacilli. However, in patients with consolidation of the lungs without bronchitis, and with cavities of which the walls are comparatively healthy, the cough and expectoration amount to very little. Generally speaking, the quantity of the sputa and the number of the bacilli are a fair test of the activity of the morbid process.

Hæmoptyses occur in a large proportion of the cases, and at any time in the course of the disease. After some of these bleedings the patients feel more comfortable, but after others there is a rapid extension of the disease. Hæmorrhages from eroded vessels in the walls of cavities are very dangerous. In some of the patients the pleurisy from time to time gives pain. In others there is a considerable exudation of serum in one of the pleural cavities, which increases the difficulty in breathing. The softening of a dead portion of the lung or the rupture of the wall of a cavity just beneath the pleura may cause perforation, pneumothorax, and then either a pleurisy with effusion or an empyema. At the time of the perforation the patients feel as if something had given way, and at once suffer from the most urgent dyspnœa. The heart's action becomes rapid and feeble and the veins throughout the body congested. The characteristic physical signs are soon evident. Such a perforation usually proves fatal within a few days or weeks.

The dyspnœa on exertion increases with the extension of the disease and the consequent diminution in the extent of lung available for breathing.

Tubercular laryngitis occurs later in the disease with chronic phthisis than with chronic miliary tuberculosis. It gives rise to cough, hoarseness, and pain.

The fever and the sweating at night seem to be related to the severity of the bronchitis and of the necrosis and suppuration of the cavities. The temperature rises and falls according to the activity of these conditions. The consolidation of the lung alone can extend with little or no fever.

The functions of the stomach and liver are sooner or later affected, either with or without chronic catarrhal gastritis and fatty infiltration of the liver. Loss of appetite, repugnance to food, nausea, vomiting, and gastric pain may seriously annoy the patient. Toward the close of the disease a severe diarrhœa often sets in, and the patients lose flesh and strength very rapidly. In these patients after death we may find tubercular ulcers of the small intestine or only a catarrhal colitis. It may also happen that extensive tubercular ulcers of the small intestine exist without any diarrhœa at all.

Tubercular meningitis or peritonitis or nephritis may occur as complicating inflammations.

Chronic degeneration of the kidney or chronic nephritis with or without exudation are often developed after the phthisis has lasted for some time.

In women menstruation is either irregular or stops altogether.

The physical signs with only a small area of lung consolidation are—dulness on percussion, broncho-vesicular or feeble breathing, increased vocal resonance, increased bronchial whisper, and in addition subcrepitant râles and friction-sounds. As more of the lung is consolidated the dulness on percussion becomes more marked. The voice and breathing approach more nearly to the bronchial character, and an increasing bronchitis gives coarse and subcrepitant râles. After cavities have been formed the percussion-sound changes to flatness, tympanitic resonance, or the cracked-pot sound. The breathing and the voice remain of bronchial quality or become cavernous. There may be gurgling râles.

The patients as a rule lose flesh and strength in proportion to the extent of lung diseased; but it is not rare to see persons who are well nourished and comparatively strong with extensive changes in the lungs.

If we compare chronic miliary tuberculosis with chronic phthisis, we may say that in the former there are but few tubercle bacilli in the sputa or in the lungs, but little necrosis or suppuration, and not much consolidation of the lung, but a loss of health and nutrition much greater than would be expected from the extent of the pulmonary lesion.

In chronic phthisis, on the other hand, the number of tubercle bacilli in the sputa and in the lungs is large, necrosis and suppuration are usually present, the patients seem to suffer rather from septic than from tubercular infection; the severity of the symptoms is usually in direct relation with the extent of lung inflamed.

Prognosis.—It is possible for the inflammatory and necrotic processes which belong to chronic phthisis to cease at any time. When this happens the symptoms of pulmonary disease may also cease, and the patients are apparently cured. The portions of lung, however, which have been destroyed or converted into fibrous tissue are never replaced by lung-tissue, so that the injury inflicted on the lungs is a permanent one. The fibrous tissue and cheesy masses left behind after the subsidence of active changes are liable to act as foci from which fresh attacks of inflammation and fresh infection may proceed. The prognosis is therefore unfavorable, although life may be prolonged in comparative comfort for many years.

Treatment.—Cases of chronic phthisis are to be managed in the same way as are the cases of chronic miliary tuberculosis.

SYPHILITIC PNEUMONIA.

In children who have inherited syphilis a number of inflammations of different parts of the body are liable to be developed soon after birth. The lungs are not exempt. They may be studded with gummy tumors of different sizes, or they may be the seat of interstitial or of intra-alveolar pneumonia.

The interstitial pneumonia may cause the consolidation of one or more lobes. The section of such a consolidated lobe is smooth and of a grayish or white color. The consolidation is effected by a growth of new tissue in the walls of the air-spaces, the bronchi, and the blood-vessels, together with the filling of some of the air-spaces with epithelium.

The intra-alveolar pneumonia also produces a whitish hepatization of considerable portions of the lung. The consolidation, however, is effected entirely by the filling of the air-spaces with fatty epithelium.

The clinical symptoms in these children are obscure. They often have other syphilitic lesions besides those in the lungs, and become weaker and more emaciated from day to day without any pulmonary symptoms except the physical signs of the consolidation.

In adults syphilitic inflammations of the lungs are rare. The best collection of cases that I have seen is that given by Hiller in the *Charité Annalen* for 1884. He gives fifty-eight cases of unmistakable syphilitic pneumonia.

Morbid Anatomy.—The inflammation is of productive character, with the formation of round-celled tissue, of gummy tumors, and of new connective tissue. There may also be some exudation into the air-spaces with a formation of epithelial cells. The new tissue is formed in the walls of the bronchi, the walls of the air-spaces, the walls of the blood-vessels, the septa between the lobules, and the pleura. It is of low vitality and may become necrotic. There results from such an inflammation, therefore, stenosis, ulceration or dilatation of the bronchi, consolidation of parts of the lungs, obliteration of the blood-vessels, lobulation of the lung, and thickening of the pleura.

The gross appearance of the lungs will accordingly vary in different cases:

1. There is an interstitial inflammation beginning at the root of the lung around the large bronchi and blood-vessels. This causes stenosis of the bronchi, consolidation of the lung, or masses of fibrous tissue along the lines of the

bronchi. The cases vary as to whether the stenosis of the bronchi or the consolidation of the lung is the principal feature.

2. The inflammation follows the type of a broncho-pneumonia, with thickening of the walls of the bronchi and small zones of peribronchial pneumonia.

3. There are large or small irregular masses or bands of dense fibrous tissue in any part of the lung. These replace the lung-tissue, and in them may be cavities formed by the dilatation of the bronchi.

4. With the interstitial pneumonia there may be associated the formation of gummy tumors or an obliterating endarteritis with areas of necrosis.

5. With more or less interstitial pneumonia at the roots of the lungs there is a syphilitic inflammation of the walls of the large bronchi, the trachea, and the larynx. The walls are thickened in some places, ulcerated in others, so that in some places there is stenosis, in others dilatation.

6. We also occasionally meet with pneumonias of the anatomical type of ordinary lobar or broncho-pneumonia; but the clinical history, although acute, is irregular, and it is probable, although not at all certain, that they are caused or modified by the syphilitic poison.

Symptoms.—Syphilitic pneumonia is one of the later manifestations of syphilis, in most of the cases coming on several years after the initial lesion. A great many of the patients have other syphilitic lesions—a fact of much assistance in making a diagnosis, a matter that is always difficult in these cases.

Of the pulmonary symptoms perhaps the most constant is dyspnœa. This is like any dyspnœa due to narrowing of the trachea or large bronchi. First, a dyspnœa on exertion; then a constant dyspnœa, made worse by the least bodily or mental exertion, and becoming more and more distressing up to the time of the patient's death.

Cough is present at some time in most of the cases—a dry cough, a laryngeal cough, or a cough with mucous or muco-purulent expectoration. Small hæmoptyses occur from time to time in some cases.

Pain referred to some part of the chest is present in some cases, absent in others.

A febrile movement seems to be the exception rather than the rule.

If a syphilitic laryngitis exists, the symptoms belonging to this will be added to those of the pneumonia.

The physical signs vary with the exact condition of the lungs, and are often obscure. They depend upon the pleurisy, the bronchitis, the stenosis or dilatation of the bronchi, and the consolidation of the lung. In the different cases, therefore, we may get tubular breathing over one or both lungs, absence of breathing over one lung or part of a lung, subcrepitant, coarse, or gurgling râles, usually localized, dulness on percussion, and increase in vocal resonance according to the extent of the consolidation.

The fact that this inflammation involves the roots rather than their apices causes the physical signs to be heard largely over the central portions of the lungs, while in tubercular inflammations it is over the upper lobes that the physical signs are usually heard.

The symptoms continue for weeks or months; the patients gradually lose flesh and strength, and finally die from the interference with breathing or worn out with the disease.

The **diagnosis** is apt to be difficult. The symptoms resemble those of chronic tuberculosis, of aneurism of the aorta, of intrathoracic tumors, and of actinomycosis of the lung. We are very dependent upon the history of the patient and the presence of other syphilitic lesions.

Treatment.—It is natural in these patients to adopt an energetic treatment with mercury and the iodide of potash, although the rule seems to be that the disease is fatal.

GANGRENE OF THE LUNG.

Definition.—Death of a portion of the lung accompanied by putrefaction.

Etiology.—Whenever the vitality of a portion of the lung is impaired, and at the same time the bacteria of putrefaction are present, there may be gangrene. It is found, therefore, with lobar pneumonia, hæmorrhagic infarctions, compression or embolism of the pulmonary or bronchial vessels, wounds of the lung, contusions of the chest, cavities in the lung, foreign bodies in the bronchi, cancer of the œsophagus, and in persons whose health has been enfeebled by disease or privation. It also occurs without discoverable cause in persons who have been in good health.

Morbid Anatomy.—Gangrene of the lung is either circumscribed or diffuse.

Circumscribed gangrene occurs in the form of one or more foci of small size where the lung is of blackish or greenish color, soft or broken down into ragged cavities. These foci have a most offensive odor. The lung around them is inflamed, and the air-spaces contain epithelium, fibrin, and pus. Such gangrenous foci, when once formed, may increase in size; as they do so the adjoining veins may become filled with infectious thrombi or eroded. From the thrombi infectious emboli can be carried into the circulation and set up inflammatory foci in different parts of the body. From the eroded vessels there are considerable hæmorrhages. If the spot of gangrene be near the pleura, it may set up either a simple or a gangrenous pleurisy, or the pulmonary pleura may be perforated and pyo-pneumothorax result. Intense bronchitis, either catarrhal or croupous, may be excited by the irritation of the gangrenous matter. If the patients recover, the gangrenous portion of the lungs is entirely removed; and a cavity is formed, the walls of which are changed into connective tissue. Such a cavity may remain for a long time or it may become contracted.

Diffuse gangrene may be secondary to the circumscribed form or it may be diffuse from the first. The greater portion of a lobe, a whole lobe, or even a whole lung, may be involved. The portion of lung involved is changed into a soft, foul-smelling, blackish or greenish mass.

Symptoms.—The patients have a cough with more or less fetid expectoration and a fetid breath. There is much variety as to the quantity of the expectoration; it may be scanty or abundant. When it is abundant and is allowed to stand for a time in a glass dish, it separates into three layers: the upper, frothy, opaque, and of a dirty-gray or yellowish color; the middle, clear and watery; the lower, greenish and purulent or mixed with blood. It consists of serum, mucus, pus, and shreds of lung-tissue. There may be, however, only a very fetid breath without any expectoration. If the pulmonary vessels are eroded, large quantities of blood are coughed up.

The patients have an irregular fever; they lose flesh and strength and pass into the septic condition. Some of the patients, however, who apparently have gangrene of the lung and recover, are not at any time as sick as one would expect.

The physical signs are sometimes obscure, sometimes well marked. They are most commonly found over the middle of the chest behind. At this point we may get dulness or flatness on percussion; absence of breathing, bronchial breathing, or cavernous breathing; exaggerated or bronchial voice and coarse râles.

If the pleura be inflamed or perforated, producing pneumothorax, the symptoms belonging to that condition are added.

The **diagnosis** is often difficult. It may be evident that the patient has a serious disease, and yet impossible for some time to determine its character. Even when it is certain that there is a pulmonary lesion, we cannot always tell whether this be gangrene, abscess, or fetid bronchitis.

The **prognosis** is always serious, but recovery is by no means impossible.

Treatment.—Besides the employment of such measures as add to the comfort of the patients and contribute to their nutrition, it is customary to give creasote or carbolic acid, either by inhalation or by the mouth. Perhaps the simplest plan is to use a Robinson's inhaler moistened with equal parts of creasote, alcohol, and chloroform.

In a moderate number of cases gangrenous cavities in the lungs have been opened and drained.

ASTHMA.

Definition.—An affection characterized by paroxysmal dyspnœa recurring at intervals, generally in the night, the dyspnœa due to a contraction of the bronchi.

The same name of asthma is also frequently employed to designate the paroxysmal dyspnœa caused by disease of the heart and by contraction of the arteries.

Etiology.—Bronchial asthma occurs most frequently in persons who have pulmonary emphysema, but it is by no means rare in persons whose lungs are normal except for the condition of the bronchi.

The causes which produce an attack of asthma may act directly on the mucous membrane of the bronchi, or indirectly on the bronchi through the blood or the nervous system. So we find some persons who never have an attack of asthma except when exposed to a directly exciting cause, while other persons have constantly recurring attacks for long periods of time without any direct cause for each attack.

Among the direct causes we reckon bronchitis, inflammations and obstruction of the nose, climatic influences, dust, vegetable irritants, chemical vapors, and animal emanations. The ordinary dust floating in the air, the odor or pollen of many plants and grasses, the vapors of pitch, sulphur, or phosphorus, the peculiar smell of dogs, cats, or horses, are familiar examples of direct causes.

The effect of climate in causing asthma is very marked in some persons. This effect does not follow any definite law, but only the idiosyncrasy of the individual. It does not matter whether the locality be warm or cold, wet or dry, low or elevated. For nearly every asthmatic person we can find some one place where he will have little or no asthma.

Of all the causes of asthma, however, bronchitis is the most frequent. In the patients who belong to this class the bronchitis constitutes the important part of the case, for if the attacks of bronchitis can be prevented there is no more asthma.

Of late years much attention has been called to diseases of the nasal passages as a cause of asthma. I think there is no question that they do constitute one of the causes; but it is going a great deal too far to say that they are the only cause.

Among the indirect causes of asthma we enumerate mental emotions, indigestion, hysteria, gout, heredity, and some of the skin diseases. It must be confessed, however, that we are often unable to say why a previously healthy person should at some particular time, without any exciting cause, begin to have attacks of asthma.

Morbid Anatomy.—As asthma is a functional disease, it has no lesions; but in the bodies of old asthmatics we commonly find the morbid changes belonging to pulmonary emphysema and bronchitis.

Symptoms.—A paroxysm of asthma begins with a feeling of oppression or suffocation about the upper part of the chest, which obliges the patient to sit up in order to breathe. The feeling of suffocation continues, and the patients bring into play all the muscles of respiration in order to satisfy the hunger for air. The skin becomes livid, the pulse feeble, and the patient's face shows his suffering. If we listen to the chest, we hear over both lungs the sibilant and sonorous breathing caused by the contraction of the bronchi. Such an attack lasts for hours or days. During the most severe attacks the patients look as if they might die at any minute, but yet after a time the attack always subsides.

As attacks of asthma are due to a variety of causes, so they present themselves to us under a variety of clinical aspects:

1. There are persons who never have asthma unless they have an attack of acute bronchitis. In such persons we have to look upon the bronchitis as the real disease, while the asthma is only a complication.

2. There are persons who only have asthma at certain times of the year and in certain localities, the attacks being caused by the inhalation of the pollen or odor of plants. These persons are said to suffer from "hay fever," "rose cold," "autumnal catarrh," etc.

3. There are persons in whom the asthma only constitutes one of the symptoms of pulmonary emphysema. These cases will be described with the latter disease.

4. There are cases in which the asthma occurs by itself as a pure neurosis. In these persons the disease is apt to be very tenacious, the paroxysms recurring again and again, even after considerable intervals of improvement. In the more severe cases the bronchi are somewhat contracted and the breathing labored nearly all the time, while the spasmodic dyspnœa recurs at regular intervals. The patients become worn out by the constant dyspnœa, the expression is one of suffering, the chest is bent forward and stooping, the nutrition is impaired, the whole condition is one of chronic invalidism, but yet life is prolonged and death is usually due to some other disease.

Treatment.—The objects of treatment are to cut short the attacks of asthma and to prevent the recurrence of subsequent attacks. To cut short an attack of spasmodic asthma we employ such means as will relax the spasmodic contraction of the walls of the bronchi. This can be done in a variety of ways—inhalation of the fumes of stramonium, nitrate of potash, chloroform, ether, or nitrite of amyl; hypodermic injections of morphine, or chloral hydrate given by the mouth or the rectum; the use of the drugs which increase the production of mucus from the bronchi, such as lobelia and grindelia robusta.

To prevent the recurrence of the attacks we examine into the condition of the nasal passages to see whether there be disease there which may cause the asthma. We inquire into the history to determine whether the asthma be not caused by bronchitis or by the pollen or odor of plants.

If the asthma be a pure neurosis, a considerable number of patients can find a climate in which they cease to suffer. The selection of this climate, however, has to be made experimentally by each patient. There is no rule to guide us. Each person has to travel from place to place until he finds the particular spot where he ceases to have asthma.

For the patients who cannot travel the most efficient treatment seems to be the long-continued administration of the iodide of potash, the systematic inhalation of compressed air, attention to the feeding, to disorders of digestion, and to any conditions which impair the general health.

HÆMOPTYSIS.

Blood which is coughed up comes for the most part from the bronchi; less frequently from the pharynx, from eroded vessels in the walls of cavities, or from aneurisms of the pulmonary artery or of the aorta.

Hæmoptysis occurs so frequently with the tubercular inflammations of the lungs, and so much less frequently with other morbid conditions, that any expectoration of blood is regarded with a good deal of suspicion.

In early tubercular inflammations of the lungs the hæmorrhage is from the mucous membrane of the bronchi. The quantity of blood coughed up may be large or small. The bleeding may last only a few minutes or may continue during a number of days. The same patient may have only a single hæmorrhage or several of them. There seems to be no period in the course of tubercular inflammation of the lungs which is exempt from the liability to bleeding from the mucous membrane of the bronchi. Especially is it to be remembered that either large or small hæmoptyses may precede by a considerble interval of time any rational symptoms or physical signs of pulmonary disease.

In the older cases of pulmonary tuberculosis in which cavities have been formed the vessels in the walls of these cavities may be eroded, with an escape of blood which is large and dangerous.

The very frequency of the association of hæmoptysis with pulmonary tuberculosis makes it important to enumerate the other conditions under which hæmoptysis may occur. The following are the forms of hæmoptysis which occur without pulmonary tuberculosis:

1. A person has one attack of hæmoptysis, only lasting for a short time, but during which he raises a considerable quantity of blood. He has no other attacks and does not develop any pulmonary lesions. The bleeding may follow severe muscular exertion, great mental excitement, or occur without discoverable cause.

2. In women hæmoptysis may take the place of menstruation. Flint says that he has seen hæmoptysis occurring at regular intervals for four years after the suspension of the menses. These cases must, however, be looked on with suspicion. A woman may cough up blood on several occasions instead of menstruating, and then develop lung disease.

3. Chronic naso-pharyngeal catarrh is sometimes attended with occasional small losses of blood.

4. There are some women who are hysterical, anæmic, always suffering from some real or fancied ailment, who from time to time cough up a little blood.

5. It is said that pregnant and nursing women sometimes have hæmoptyses.

6. With disease of the aortic and mitral valves, especially with mitral stenosis, bleeding from the bronchi is of frequent occurrence. In the course of the heart disease the patients from time to time, during periods of several days, cough up clear blood in considerable quantities.

7. Aneurisms of the branches of the pulmonary artery within the lungs, when they rupture, cause fatal hæmorrhage, a large part of the blood being coughed up.

Aneurisms of the arch of the aorta which erode the trachea or main bronchi may rupture into these tubes by small or large openings. With the small openings the patients cough up a little blood from time to time, while part of the escaped blood is inspired into the lungs and sets up a peculiar form of pneumonia. With the large openings the blood escapes through the trachea in enormous quantities, and the patients bleed to death within a few minutes.

8. Patients who suffer from emphysema and chronic bronchitis not infrequently from time to time cough up small quantities of blood. Much less often such patients have a large bleeding from the bronchi. Part of this blood is coughed up at once, part is coagulated in the large bronchi, and is afterward coughed up in the form of casts of these tubes.

9. Sir Andrew Clark[1] describes a form of bleeding which he calls "arthritic hæmoptysis," of which he says that he has seen some twenty cases. He lays down the following propositions: There occurs in elderly persons, free from ordinary diseases of the heart and lungs, a form of hæmoptysis arising out of minute structural alterations in the terminal blood-vessels of the lung. These vascular alterations occur in persons of the arthritic diathesis, resemble the vascular alterations found in osteo-arthritic articulations, and are themselves of an arthritic nature. Although sometimes leading to a fatal issue, this variety of hæmoptysis usually subsides without the supervention of any coarse anatomical lesion of either the heart or the lungs. This variety of hæmorrhage is aggravated or maintained by the frequent administration of large doses of strong astringents, and by an unrestricted indulgence in liquids to allay the thirst which the astringents create. The treatment which appears to be the most successful in this variety of hæmoptysis consists of diet and quiet, in the restricted use of liquids, and the stilling of cough; in calomel and salines, in the use of alkalies and iodide of potassium, and in frequently renewed counter-irritation.

10. Dr. Flint[2] says that he has met with a few cases of persistent bronchial hæmorrhage. In two of these cases, after expectorating daily more or less of a sero-sanguinolent liquid during several months, recovery took place under the use of tonics and hygienic measures. In the third case the hæmorrhagic expectoration continued for six years, during which time repeated examinations of the chest failed to discover any positive signs of pulmonary disease.

11. Severe injuries inflicted upon the wall of the thorax may be followed by the expectoration of blood for hours or days.

Treatment.—In managing cases of bleeding from the mucous membrane of the bronchi it is important to bear in mind that even the most profuse hæmorrhages are seldom fatal.

The methods of treatment commonly employed are—the application of cold to the chest, the temporary ligation of one of the arms or legs, the internal use of opium, ergot, hydrastis, krameria, tannic acid, gallic acid, acetate of lead, persulphate or pernitrate of iron, or of calomel, or of the saline cathartics. It is also customary to keep the patients very quiet while the bleeding is going on. I doubt if it be wise to be too anxious and energetic in the treatment of bleeding from the mucous membrane of the bronchi. The bleeding, as a rule, will stop, no matter what is done. The frequent use of astringents disorders the stomach, the insistence of absolute quiet demoralizes the patients, keeping the patients on a low diet unnecessarily reduces their strength.

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[1] *Lancet*, 1889, p. 841.

[2] *Practice of Medicine*, p. 265.

INTERLOBULAR EMPHYSEMA.

Definition.—An accumulation of air in the connective-tissue septa between the lobules of the lung.

Etiology.—Interlobular emphysema is, I think, most frequently seen with the broncho-pneumonia of young children. It may be caused by any violent efforts which produce the abrupt introduction of a large quantity of air into the lungs and its forcible retention therein by closure of the glottis. The efforts in parturition, defecation, raising weights, coughing, paroxysms of rage, excessive laughter, and hysterical convulsions have all been occasionally followed by rupture of the air-cells and interlobular emphysema.

Morbid Anatomy.—We find after death the interlobular septa infiltrated with air, with more or less compression of the parenchyma of the lung. The air may find its way from the lung into the mediastinum, and thence into the connective tissue of the neck and the wall of the thorax.

Symptoms.—There seems to be no distinctive rational or physical symptoms belonging to interlobular emphysema; but in extreme cases it is said to have caused sudden death.

VESICULAR EMPHYSEMA.

It is customary to speak of three forms of vesicular emphysema: Compensating Emphysema, Senile Emphysema, and Substantive Emphysema.

1. COMPENSATING EMPHYSEMA.—If one lung or a part of one lung be so changed by disease that it can only partially perform its functions, the other lung becomes increased in size and its air-spaces are dilated. This change in the lung is a beneficial rather than a morbid one, and gives no symptoms of disease.

2. SENILE EMPHYSEMA.—This condition is often described as something different from substantive emphysema. It is said that, instead of there being an abnormal accumulation of air in the lungs, these organs are smaller and contain less air than normal. In consequence of atrophy of the alveolar walls the air-cells coalesce and form larger air-spaces. These, however, do not result from a dilatation of the alveoli, but from a gradual shrinkage and disappearance of the lung-tissue.

I must confess that such descriptions of senile emphysema do not correspond with the lungs which I have seen. I believe that the anatomical conditions are the same in senile as in substantive emphysema, although the causation and clinical history are different.

3. SUBSTANTIVE EMPHYSEMA.—The definition of substantive emphysema

usually given is that it is a morbid condition of the lungs characterized by enlarged capacity of the air-cells, with atrophy of their walls and obliteration of their capillaries. I should define the disease, on the contrary, as a chronic interstitial inflammation of the lungs, with which more or less dilatation of the air-spaces is associated.

Etiology.—Laennec, who was the first to describe this lesion, gives the causation of emphysema as follows: Chronic catarrh, plugging by mucus of small bronchi, consequent obstruction to the passage of air, conveyance by inspiration of air past the obstruction, failure of expiration to expel this air, accumulation of air in the air-spaces, dilatation of the air-spaces. The foundation of the disease, therefore, is bronchitis.

Louis denies this mechanism, because the symptoms of dilated air-cells are not preceded by catarrh, because habitual dyspnœa does not undergo permanent increase after acute catarrh, and because normal inspiration is not more powerful than expiration.

Dr. Williams maintains the catarrhal basis of Laennec, but supposes that, while the air-cells communicating with plugged bronchi escape distension, those adjoining and possessed of free communication with the trachea dilate in consequence of the extra work and pressure thrown upon them.

Walshe says that the vesicular dilatation may be a primary or a secondary phenomenon; that is, it may occur independently of any acknowledged form of statical change within the chest or it may supervene on some actual organic mischief. The dilatation may be the resultant of primary nutritive change in the actual walls of the enlarged vesicles, affecting both their statical and dynamic properties. Or these cells, being in their own nature healthy, may dilate through the extra strain thrown on them in consequence of the inaction of neighboring portions of lung.

It is frequently stated that playing on wind instruments or glass-blowing causes emphysema.

If, on the other hand, we believe that emphysema is a chronic interstitial inflammation of the lung, and that the dilatation of the air-spaces is not the primary or essential part of the morbid process, then we class emphysema with chronic endocarditis, endarteritis, and nephritis, and ascribe it to the same causes.

It is generally agreed that the disease is of more common occurrence in some families than in others.

The tradition has been handed down from one text-book to another that persons who have emphysema are less liable than are others to have tuberculosis of the lungs or lobar pneumonia. This tradition seems to have no foundation in fact. Tuberculosis and emphysema of the lungs are frequently associated.

Morbid Anatomy.—Both lungs are as a rule increased in size. The dilatation of the air-spaces may be so great as to be evident to the naked eye or so moderate as not to be appreciable. When we examine minutely the lungs of a large number of persons who have, during life, suffered from the symptoms of emphysema, we find a very great variety in the morbid conditions. In some both the air-passages and air-vesicles are largely dilated; in some the air-passages alone are dilated, the air-vesicles remaining of normal size; in some neither the air-passages nor the vesicles are appreciably dilated. We find also, when we compare the lungs with the clinical histories which belong to them, that the most marked symptoms are often associated with very slight degrees of dilatation of the air-spaces, and it is evident that the severity of an emphysema is not to be measured by the degree of dilatation of the air-spaces.

The walls of the air-spaces are thickened in some parts of the lung, thinned in others. The epithelial cells which line the walls of the air-spaces are often increased in size and number. In the walls of the air-spaces are holes with sharp-cut edges. These holes are formed in the spaces between the capillaries: some are very minute, others are of large size. It is evident that these holes are not formed by the stretching of the air-spaces, for they are found in small air-spaces as well as in large ones. They constitute a curious and important part of the lesion. The septa between the lobules, the connective tissue around the bronchi and blood-vessels, and the pulmonary pleura are often considerably thickened. Very frequently there are adhesions between the pulmonary and costal pleura. The mucous membrane of the larger bronchi is often thickened.

The blood-vessels of these lungs can be readily filled with an artificial injection. Neither arteries, capillaries, nor veins are obstructed or obliterated; but in the walls of dilated air-spaces, and in the walls of those in which there are holes, the meshes of the capillary plexus are larger and the capillaries are farther apart from each other.

During life, however, in some cases of emphysema there is an obstruction to the passage of blood through the lungs, and consequently dilatations and hypertrophy of the right ventricle of the heart and venous congestion of the pia mater, stomach, small intestine, liver, spleen, and kidneys. These evidences of venous congestion often exist in cases in which the air-spaces are but very little dilated.

It is not often that we see a case of advanced emphysema after death without finding at the same time chronic endarteritis, endocarditis, or nephritis.

Symptoms.—*Physical Signs.*—In the lesser degrees of emphysema there is no change in the shape of the thorax. In the more advanced cases there is a prominence of the upper part of the sternum and of the costal cartilages. In patients who have suffered much from dyspnœa the hypertrophy of the muscles which move the thorax contrasts with the general emaciation of the patients. In the cases in which there is great dilatation of the air-spaces the chest assumes the so-called barrel shape.

The percussion sound may remain unchanged for a considerable length of time. When it is changed, the change is either to a rather dull note of wooden quality or to exaggerated resonance of either vesicular or vesiculo-tympanitic quality. The respiratory murmur is feeble or there is feeble inspiration with longer, louder, low-pitched expiration; or both inspiration and expiration may be exaggerated, loud, and high-pitched.

The pleuritic adhesions give more or less dulness on percussion. When the bronchi are contracted there is sibilant and sonorous breathing.

Rational Symptoms.—There are many persons in whom substantive emphysema is developed and continues for many years without giving rise to any symptoms, and yet even in such persons it is often possible to be pretty sure of the presence of the disease, because they are persons whose general condition and age are such as are usually associated with emphysema.

There are many persons in whom the associated chronic endocarditis or endarteritis or nephritis gives such marked symptoms that the emphysema passes unnoticed.

In some persons the emphysema produces after a time dyspnœa on exertion, but without bronchitis or disturbance of the general health. These are persons past middle age, who do not consider themselves invalids, who, on the contrary, are often strong and robust, and in whom the emphysema is only an inconvenience.

In some persons the principal symptoms are those belonging to the associated acute and chronic bronchitis. The attacks of acute bronchitis may be mild, lasting for a few days or a few weeks, with cough, mucous expectoration, sometimes hæmoptyses, asthmatic breathing, and a febrile movement; or they may be severe and last for several months with continued cough, asthmatic breathing, fever, venous congestion, dropsy, and loss of flesh and strength. The chronic bronchitis continues year after year, better every summer and worse every winter. The patients have a cough with mucous or muco-purulent expectoration, sometimes with small hæmoptyses. They are always a little short of breath when they exert themselves. After a time they have attacks of spasmodic asthma. In the unfavorable cases the dyspnœa on exertion becomes more constant and more decided, venous congestion and dropsy are established, and the patients lose flesh and strength.

In some persons the prominent feature in the disease is the liability to attacks of spasmodic asthma, which often are frequently repeated and of long duration. These attacks are sometimes due to contractions of the bronchi, and then we get sibilant and sonorous breathing; or they are due to contraction of the arteries belonging to the aortic system, and there is a radial pulse of increased tension; or they are due to contraction of the branches of the pulmonary artery, and then there is neither sibilant and sonorous breathing nor a pulse of high tension.

In some persons the principal symptom is the constant dyspnœa. The difficult breathing is at first only developed by exertion; later it becomes more constant, and is made worse by slight exertion, by indigestion, and by bronchitis. Finally, in the bad cases the dyspnœa is constant and distressing. The patients constantly feel the need of air, and are always over-using the muscles of respiration in order to satisfy this need. General venous congestion is gradually established, as well as cyanosis of the skin, clubbing of the fingers, congestion of the stomach, small intestine, liver, and kidneys, dilatation and hypertrophy of the right ventricle of the heart, and general dropsy. The nutrition of the patients suffers, and they become emaciated, feeble, and anæmic. It is not easy to tell how much of this dyspnœa depends upon the anatomical changes in the lungs, how much upon contraction of the branches of the pulmonary artery, and how much upon contraction of the arteries belonging to the aortic system.

There are rare and fatal cases in which there are no pulmonary symptoms. The patients lose flesh and strength and become anæmic without any evident cause for their ill health. After continuing in this way for some time, they begin to have attacks of contraction of the arteries, with headaches, sleeplessness, delirium, stupor, muscular twitchings, and vomiting, or a dyspnœa like that seen with chronic nephritis. They die within a few months after they have begun to have the attacks of contraction of the arteries.

In some persons, after emphysema has existed for some time with more or less marked symptoms, chronic miliary tuberculosis is slowly established.

Emphysema by itself proves fatal only in a moderate number of cases. Death is usually due to some complicating or intercurrent disease.

Treatment.—The conditions which call for treatment are the morbid condition of the lung, the loss of nutrition, the bronchitis, the constant dyspnœa and the spasmodic dyspnœa, the contraction of the arteries, and the venous congestion.

The emphysema is favorably affected by an out-of-door life in a suitable climate; by abstinence from alcohol, tobacco, sugars, and starches; by the use of fats and by over-feeding with the stomach-tube; and by methodical inhalations of compressed air. All of these measures are also of service in improving the nutrition of the patients and in helping them to get rid of chronic bronchitis.

The constant dyspnœa is due to the changes in the lungs, and is then to be treated by the same means which are used to control the emphysema, or it is due to the complicating contraction of the arteries, and is then to be treated by the drugs which dilate the arteries—nitro-glycerin, potassium iodide, or chloral hydrate.

The attacks of spasmodic dyspnœa are due to—

(*a*) Spasmodic contraction of the muscular coat of the bronchi. This can be relieved by the inhalation of the fumes of stramonium, nitrate of potash, chloroform, or ether—by the administration of chloral hydrate, potassium iodide, belladonna, or opium.

(*b*) Congestion of the walls of the bronchi. This can be relieved by drugs which increase the production of mucus, such as lobelia and grindelia robusta; by drugs which stimulate the heart, such as caffeine, convallaria, and digitalis; or by the application of dry cups to the walls of the chest.

(*c*) Contraction of the small arteries. This can be relieved by the drugs which dilate the arteries, such as nitrite of amyl, nitro-glycerin, chloral hydrate, potassium iodide, or opium.

(*d*) Inflammation of the nasal passages. This is to be treated by local applications.

ACTINOMYCOSIS OF THE LUNG.

The following account of actinomycosis of the lungs is taken from a compilation of thirty-four cases made by Dr. Hodenpyl; my own experience is limited to two cases.

Definition.—Pulmonary actinomycosis is a chronic infectious disease of the lungs dependent upon the presence of actinomyces.

Etiology.—Information concerning the characters and causation of actinomycosis in general are given in the article on that disease. So far as the lungs are concerned, the living germ seems to be inhaled into the bronchi. The majority of the cases were in young adults; the youngest patient was nine years old, the oldest sixty-three.

Morbid Anatomy.—The lesions are unilateral in about the proportion of three to one. They may be classified into two groups:

1. There are cases with the symptoms of chronic general bronchitis, with the germ present in the sputum, but in which no autopsy is made. Whether in these cases there is no lesion but that of chronic bronchitis we cannot certainly tell.

2. There is a broncho-pneumonia of a peculiar type which involves part of a lobe or an entire lung. The large bronchi are coated with muco-pus. The small bronchi contain pus, their walls are thickened, they are surrounded by zones of peribronchial pneumonia. In some of the small bronchi there are growths of new connective tissue partly filling them. In the peribronchial zones of pneumonia the walls of the air-spaces are thickened and their cavities filled with new connective tissue. Between these zones is a diffuse hepatization of ordinary exudative type. There are adhesions between the pulmonary and costal pleura. There are often, in addition, sacculated collections of pus in the pleural cavity, which may perforate through the skin or through the diaphragm. The ribs, sternum, or vertebræ may be eroded. The opposite lung, the pericardium, or the heart may become involved. There may be secondary inflammations of the abdominal organs or of the brain. In one case the inflammation penetrated the portal vein, and there were metastases all through the body.

Symptoms.—A febrile movement is present in nearly all the cases. Usually it is one of the first symptoms, but sometimes it does not come on until later in the course of the disease.

Cough is usually the first symptom and continues throughout the disease. It is accompanied with an abundant muco-purulent, often fetid, expectoration, and sometimes contains actinomyces. Hæmoptyses were not observed, although the sputa were sometimes stained with blood.

The patients lose flesh and strength, at first slowly; later, as abscesses are formed and septic poisoning established, they run down more rapidly.

The physical signs are those of bronchitis, of broncho-pneumonia, of phthisis, or of empyema.

The average duration of the disease is ten months; the shortest case lasted four months, and the longest was still living after a duration of several years. Of the thirty-four cases, all died except two.

Diagnosis.—The disease is liable to be confounded with fetid bronchitis, gangrene of the lung, broncho-pneumonia, or pulmonary phthisis. The only positive diagnostic symptom seems to be the presence of the actinomyces in the sputa or in the pus from the pleura.

Treatment.—There seems to be no way of directly improving this disease of the lungs.

MALIGNANT GROWTHS IN THE LUNGS.

We include under this head the primary and secondary tumors formed in the lungs which belong to the classes of carcinoma, sarcoma, and lymphoma.

Morbid Anatomy.—The carcinomata of the lung are either secondary or primary. The secondary tumors follow the anatomical type of the primary tumor. The primary tumors consist of a stroma enclosing spaces lined with cylindrical epithelium, the growth apparently beginning in the small bronchi. Whether the tumors are primary or secondary, we find that they may be developed in such a way as to compress the bronchi; or be associated with exudative and productive inflammation in such a way as to consolidate large portions of the lung; or be associated with suppurative and destructive inflammation in such a way that abscesses are formed; or involve the pleura so that large collections of serum are formed in the pleural cavities.

The sarcomata of the lung are secondary tumors. They usually are in the form of nodules scattered through the lung, or of tumors which compress the bronchi, or of tumors in the pleura with serum in the pleural cavities.

The lymphomata begin in the bronchial glands and infiltrate the lungs from the root outward, following the track of the bronchi or of the interlobular septa.

Symptoms.—While in some cases the symptoms are obscure, in others they are well defined. They are apt to follow one of three types:

1. The most marked symptom is dyspnœa, due to pressure on the bronchi. The dyspnœa is developed slowly and is brought on by exertion. It becomes more and more distressing, until finally the patient can hardly move at all without bringing on the difficulty in breathing.

The physical signs are either tubular breathing or diminished breathing over the lung corresponding to the compressed bronchi. The patients lose flesh and strength, at first slowly, later very rapidly.

2. The symptoms are those of a chronic inflammation of the lung. The patients have cough, dyspnœa, muco-purulent or bloody expectoration, a

febrile movement, and pain in the chest, the physical signs of bronchitis and of consolidation of the lungs, and gradual loss of flesh and strength.

3. The symptoms are those of a pleurisy with effusion, but it is a pleurisy which does not improve under treatment. The serum is apt to be blood-stained, but is not always so; it reaccumulates after it has been drawn off: the patients steadily lose flesh and strength.

PRACTICAL URINARY EXAMINATION.

By J. W. HOLLAND.

The Ordinary Examination.—It does not fall within the scope of this article to consider the physiological, nor even all the pathological, relations of the urine. As it is concerned with the knowledge which has value to the practitioner, it is deemed best to limit the range of inquiry to those points which have practical significance.

A good working plan for the ordinary analysis need not include more than the following procedures, and in most cases less than the total of these will serve every requirement:

1. Measurement of the daily quantity.
2. Noting the color: if deep yellow, green, or brown, testing for biliary pigment; if reddish, smoky, or chocolate-hued, testing for hæmoglobin.
3. Taking the reaction.
4. Determination of specific gravity with the hydrometer.
5. After the sediment falls, decanting the clear part and examining for albumin by boiling and by adding acid—nitric, picric, or acetic. If greenish flakes form, bile-pigment to be looked for; if red-brown, then hæmoglobin. After twenty-four hours noting the height of albuminous layer.
6. Testing for glucose by Fehling's and by Böttger's methods, with calculation of the amount.
7. Estimation of the relative amount of chlorides.
8. Estimation of the amount of urea.
9. Noting the naked-eye appearance of the deposit which forms on standing for several hours. Making allowance for the light cloud of epithelial débris sometimes found in health, a sample voided turbid and acid points to urates or mucus or pus or blood; if voided turbid and alkaline, points to phosphates.
10. Careful examination of the deposit with the microscope, using a one-quarter or one-fifth objective and an eye-piece giving a magnifying power of about 250 diameters. The search should be made for phosphates, calcium oxalate, uric acid, urates, epithelium, pus, tube-casts, spermatozoids, blood-cells, leucin, tyrosin, cystin, organisms such as sarcinæ, the moulds, and bacteria; and in addition such extraneous substances as sometimes enter the bladder by fistula from the rectum.

For minute study of the bacteria use the high power of 900 diameters obtained with immersion lenses. If the absence of organic or definite crystalline structure leaves a doubt as to the nature of a deposit, the following simple tests may prove serviceable: First, warm a portion of the deposit with some urine in a test-tube: if it clears up, then the urates are present; if it does not clear up, then suspect phosphates. Second, warm a fresh portion with acetic acid: if it dissolves, phosphates are present.

Precautions as to the Sample.—The microscope often shows substances which, being extra-urinary, may be broadly described as dirt, having no significance whatever. Owing to ignorance or carelessness on the part of patient or nurse, it not infrequently happens that floating dust or sweepings or fæcal matter get into the vessel, or sometimes an unclean bottle may make its contribution. Hairs, cotton and linen fibres may be mistaken for tube-casts, while such objects as large globules of free oil, starch-granules, and vegetable cells are obviously extraneous. To avoid fallacies, it is well to enjoin care upon the patient or nurse. The urine should be voided into a well-cleaned chamber-vessel, or, better still, into a glass collecting-jar sufficiently large to hold the entire daily amount. By means of a clean glass funnel about eight fluidounces should be transferred to a bottle or, if in hospital, to a conical glass.

Before taking up a drop of the deposit with the pipette sufficient time must be allowed for the sediment to collect. As a rule, this will require that the sample should stand for about three hours. The readiness with which urine undergoes change is a noteworthy fact. The liability varies in different specimens. Even a healthy urine may in a few hours after micturition increase in acidity, owing to the change of the common soluble urates to the more acid and less soluble salts, which are precipitated along with more or less free uric acid. The destiny of the urea in all specimens kept several days in a warm place is to be converted into ammonium carbonate by the growth of the micrococcus ureæ. This change may take place in the bladder if the urine is retained too long, and may cause grave complications in vesical diseases.

The urine itself becomes turbid, putrid, and irritating, throwing down a deposit of phosphates with urate of ammonium. To correct this tendency in cases of cystitis, it is customary to wash out the bladder with a saturated solution of boric acid or some other unirritating antiferment.

Preservative Fluid.—The sample of urine should be examined within twelve hours after micturition, and preferably within three hours, merely allowing time for the deposit to settle. When it is desired to preserve a specimen for several days, it suffices to add salicylic acid, about 3 grains to the half pint of urine. This will not prevent the changes of structure which sometimes take place in blood-cells, tube-casts, and renal epithelium when the urine is of low density. To protect these from alteration the density must be raised by adding some mineral salt, such as potassium acetate, in saturated solution. According to Wendringer, a complete preservative fluid for dissolved constituents, such as albumin and sugar, as well as for organized deposits, can be made by mixing 12 parts of powdered borax, 12 parts of boric acid, and 100 of hot water. Having stirred the mixture well, it should be filtered while hot. The sample of urine should be put into a suitable glass, and one-third of its bulk of the boric solution is added and agitated with it. If the urine be turbid from urates, it becomes clear, but no other alteration occurs. Albumin is not coagulated nor are the organic deposits affected injuriously.

NORMAL URINE.—The urine of health is a clear solution in water of various substances. Some of these impart a feebly acid reaction; some give it a yellowish color; some are the source of its characteristic odor; and all combined raise its specific gravity to a point between 1015 and 1025. The proportions of these constituents are not constant for all individuals, nor even for the same person taking one day with another; indeed, they vary hourly. In making a statement of average composition regard is had to this variable character: the figures which follow may be taken as representing the average amounts in round numbers.

The chief solid constituent is urea, which is present to the extent of about 20 parts in 1000 of urine. Other solids of less importance are uric acid, 0.4; hippuric acid, 0.75; kreatinin, 0.75; pigment, mucus, xanthin, and various extractives, 10; chlorides, 10; sulphates, 1.1; alkaline phosphates, 1.2; earthy phosphates, 0.8. These are the ingredients commonly occurring in appreciable amounts.

THE QUANTITY.—In making a quantitative determination of any constituent, not only must the tested sample be a portion taken from the total mixed urine of the day, but the daily quantity of the urine itself must be known. The large collecting-jar may be graduated so as to be the measuring vessel: such wide-mouthed graduated jars as are used by druggists for percolating will serve admirably, though the common glass "specie jar" is about as good, and is easily obtained anywhere. It must be large enough to hold the entire daily discharge, and then for measuring the volume a smaller apothecary's graduated glass can be used. The wide mouth admits of the introduction of the hand for the thorough washing always required before beginning the daily collection. The patient is instructed to empty his bladder at a given hour, but not into the jar. Afterward, for twenty-four hours, the urine is passed into the one jar, which should be kept in a cool place, and at the given hour the last contents of the bladder are added to it. The amount should then be noted, and about 8 fluidounces put into a perfectly clean glass bottle or other vessel to serve as a sample for analysis.

Practical Import.—The mean daily discharge is 1250 c.c., or 50 fluidounces, or 3 pints. In drawing conclusions from any change in this respect, it is necessary first to note that even in health there may be considerable variation. The amount voided will depend, first, upon the amount of water drunk; it will be affected by the proportion of water lost in perspiration and the quantity retained in the tissues as necessary for nutrition. These factors are various in different men, and change with the season and with the habit of exercise. It is compatible with health in some for the daily discharge to reach only 1½ pints, and at times for it to go as high as 4 pints. Making allowances for these physiological variations, the urine is notably scanty in certain forms of Bright's disease, in cirrhosis of the liver, and in the state of collapse occurring in cholera or the pernicious fevers. By *anuria* is meant a condition in which the urine is no longer voided: this includes *suppression*, when the secretion of the kidney is suspended, and *retention*, when the fluid, although secreted, is retained in the urinary passages by mechanical obstruction.

A persistent excess of the aqueous constituent, without a corresponding increase of the solids, is termed *hydruria*. This symptom is characteristic of diabetes insipidus, in which disease the daily discharge may be more than 8000 c.c., or 2 gallons, while the specific gravity sinks to 1003 or less. Some diuresis occurs in the middle period of atrophic nephritis. Hysterical and neurotic subjects may suffer temporarily from a too copious urinary flow.

By *polyuria* is meant an excess not only of urinary water, but of all the solid constituents. It would include cases of "azoturia," in which the urea is morbidly abundant, and the "phosphatic diabetes" of Teissier, which accompanies an excessive tissue-waste, like that of saccharine diabetes, but in which no sugar is found.

THE COLOR.—Normal urine is amber-hued, the depth of color varying as the proportion of coloring matter varies. When the volume of urine is low, the liquid is dense and the color deepens to a reddish tint. After liberal drinking, followed by copious urination, it may be almost as colorless as water itself. Besides its true coloring principle, the urine contains sulph-indoxylate of potassium or indican, a colorless substance which forms indigo-red or indigo-blue by the action of reagents. Its presence may be shown by adding to 100 parts of normal urine 10 parts of hydrochloric acid. After standing for half an hour the amber color changes to pink from the formation of indigo-red. Diseases of the liver and bowels which cause constipation thereby favor the absorption of indol from the fæcal mass, and an increase of its derivative, indican, in the urine. This increase is revealed by the deeper color yielded when the acid test is applied. The urine is pale yellow in the free flow of diabetes and after attacks of hysteria or epilepsy; orange-red from the elimination of santonin in an alkaline medium; reddish naturally after full meals with small potations, after severe exercise with abundant sweating, during paroxysms of fever, after hæmorrhage into the genito-urinary tract, and, lastly, after the administration of logwood; brownish from the condition known as melanosis, from hæmoglobinuria, and from the administration of tar, carbolic acid, gallic acid, tannic acid, senna, or sulphonal. In jaundice the biliary coloring matter (*q. v.*) will make it sulphur-yellow or olive-green.

Practical Import.—It is plain that excess of indican would point to diseases retarding digestion or causing constipation, though it has been found in cholera and all forms of severe cachexia. The detection of foreign coloring matter would furnish indications of an obvious character based upon the nature of the specific substance: for that of blood consult the section on Hæmaturia; for that of bile, the section on Biliary Coloring Matter.

SPECIFIC GRAVITY.—When it is desired to make use of this property in determining by special formulas the amount of urea or of sugar in solution, it is best to take the observation with the specific-gravity bottle. A bottle of known capacity, say of 1000 grains, is counterpoised, then filled with urine, and weighed in a delicate balance. If the contents weigh 1025, that number will be the specific gravity. This operation requires apparatus not always at hand, and consumes time not always at the disposal of the physician. For ordinary purposes it suffices to take the specific gravity with a urinometer, which is a spindle hydrometer for liquids heavier than water, carrying a scale ranging from 1000 to 1060, and usually made to be read at 60° F. or 15° C.

Method.—Fill to one-half its capacity a cylindrical vessel having a level lip. Gently immerse the urinometer and carefully fill the cylinder to the brim. Take the observation by sighting on a level with the surface of the urine, which rises slightly above the edge of the glass.

Practical Import.—The standard of health is usually rated as between 1015 and 1025, but very free use of beverages may cause it to fall below 1010. Under ordinary conditions in regard to the amount of fluid ingested so low a density would point to diabetes simplex or to Bright's disease with deficiency of urea. When the record is above 1030, it usually denotes sugar in the urine. In either case the proper chemical tests would solve the doubt.

It is possible to derive valuable conclusions from roughly estimating the solid constituents of the urine by multiplying the last two figures of the specific gravity with Haeser's factor, 2.33. This gives parts of solids per thousand of urine, and furnishes some idea of the efficiency of the kidney at the time.

REACTION.—In noting the reaction blue and red litmus-papers may be used, but the most convenient indicator is violet-colored "neutral" litmus-paper. Acids turn it red and alkalies blue. A sample of the total daily urine of health should turn this "neutral" paper, or even the blue paper, to a delicate red. This shows an acid reaction due to the dissolved acid phosphates, and perhaps urates. Sometimes in health a sample representing the unmixed urine secreted during the first or second hour after a full meal is alkaline or neutral, as an effect of the preponderance of alkaline salts in the food. The paper made blue by such a sample retains the blue color, thus indicating "fixed alkali"—*i. e.* alkaline salts of sodium and potassium.

The same change can be produced at will by the repeated administration of large doses of the bicarbonates or citrates of sodium and potassium, as in the alkaline treatment of rheumatism. When the paper is turned blue by the "volatile alkali" ammonia, we may know it by the gradual restoration of the original color as the ammonia volatilizes. A characteristic putrid odor attends this reaction. It is due chiefly to the ammonia evolved from ammonium carbonate resulting from the decomposition of urea by the micrococcus ureæ.

$$\underset{\text{Urea.}}{CON_2H_4} + \underset{\text{Water.}}{H_2O} = \underset{\text{Ammonium carbonate.}}{(NH_4)_2CO_3}.$$

Practical Import.—Persistent alkalinity due to fixed alkali is sometimes found in persons of a feeble habit of body. The change in reaction lessens the solvent power of urine for the earthy phosphates, which in consequence are precipitated in loose whitish amorphous particles. These do not tend to form concretions. If the alkalinity be due to ammonia, the indication is very different. The ammoniacal change occurring in the bladder is a concomitant of serious vesical mischief. The earthy phosphates are mixed in a deposit with the "triple phosphates" of ammonium and magnesium. If the bladder be not kept well washed of this deposit, it will in time accrete into the "mixed phosphate" gravel or calculus. In every case of incomplete evacuation of the bladder from paralysis or obstruction this is a rock ahead.

It remains to be said that the administration of acids, unless it be benzoic acid, while tending to acidify the urine, will have little direct effect upon the reaction. The strongest acids, given in usual doses, are neutralized before they enter the circulation. Whatever power they have over the alkaline urine of feeble subjects is explained by the increased tone they impart to digestion, thus removing debility and anæmia. Given by the mouth, they exert no control over ammoniacal urine.

PHOSPHATES.—The urinary phosphates may be divided into two groups, earthy and alkaline, according to their bases. The total daily discharge of these is about 60 grains or 4 grammes, the acid phosphates of calcium and magnesium, or "earthy" $MgHPO_4+Ca_3(PO_4)_2$, constituting one-third, and the acid sodium and potassium phosphates, or "alkaline" $(NaH_2PO_4+KH_2PO_4)$, the other two-thirds. In order to estimate approximately the total phosphoric acid, resort may be had to Teissier's method. All the apparatus needed is a glass cylinder graduated in the cubic centimetres of the metric system. Put into the graduate 50 c.c. of urine, and add 15 c.c. of magnesia mixture (magnes. sulph. parts 10; ammon. chlor. 10; aq. ammon. fort. 10; water 80). Shake well and set aside for twenty-four hours. All the phosphoric acid is thrown down as ammonio-magnesium phosphate, a dense white deposit. At the end of twenty-four hours note the number of cubic centimetres occupied by this sediment. For 1 c.c. there are 0.30 grammes per litre, or 0.03 per cent. of phosphoric acid, equivalent to 0.60–0.70 grammes of phosphates per litre, or 0.06–0.07 per cent. To obtain a result in terms of grains to the fluidounce multiply by 4.55.

For more accurate calculations the volumetric method will serve. The standard solutions used and the indicators can be had of all first-class druggists, who keep formularies in which the mode of preparation is given. Put 50 c.c. of urine into a porcelain capsule, and add 5 c.c. of a saturated solution of sodium acetate containing an excess of acetic acid. Heat on a sand-bath or wire gauze until boiling begins, and then from a burette slowly add about 2 c.c. of standard solution of uranium acetate, causing a precipitate of uranium phosphate. Stir with a glass rod, and touch its wet end to a drop of solution of potassium ferrocyanide on a white plate. A red-brown color indicates that too much standard solution has been used, and the process must be repeated. However, this is not likely when only 2 c.c. have been used. If no red-brown spot appears, continue to add from burette slowly, and after the addition of each 1 c.c. stir and touch the rod to the ferrocyanide. When the red-brown spot appears read off the number of cubic centimetres taken from the burette. The standard solution contained 31.1 grammes of uranium acetate to 1000 c.c. of water, equal to 5 grammes of phosphoric acid (P_2O_5). If 1000 c.c. = 5 grammes P_2O_5, then 1 c.c. will represent 0.005 grammes P_2O_5 in the 50 c.c. of urine employed.

The terms in percentage can be obtained by multiplying the number of cubic centimetres used by 0.01, which is the equivalent of 0.005×2. To get grains to the fluidounce multiply the per cent. by 4.55.

Practical Import.—When it is considered that the phosphates of the urine are derived only in part from the waste of nervous tissue, part being supplied by the rest of the body, and an uncertain amount coming almost directly from the same principles in food, it will be easily understood why the quantitative estimates so far have proved of no direct value to the clinician. Their significance depends not on the relative proportion in the sample, but upon their state. Any amount, large or small, in an undissolved state will figure as a deposit, and thereby have pathological meaning.

PHOSPHATIC DEPOSITS.—It has been said above that the phosphates of normal acid urine are held in clear solution. When the urine is alkaline, it loses its solvent powers for the earthy phosphates, and throws them down as a grayish-white sediment made of colorless granules, which show no tendency to aggregate into masses having particular shapes.

The amorphous urates form into groups which branch somewhat like sprigs of moss. A drop of acetic acid insinuated under the cover-glass will clear away the phosphates, but not the urates. If the alkalinity be due to the ammoniacal fermentation of urea, then the ammonia reacts with the magnesium phosphate to produce the white crystalline deposit of ammonio-magnesium phosphate, the so-called "triple phosphate."

$$\underset{\text{Magnes. phosph.}}{2(MgHPO_4)} + (NH_4)_2CO_3 = H_2CO_3 + \underset{\text{Triple phosphate.}}{2(MgNH_4PO_4)}.$$

Usually the crystals are large enough to be seen by the naked eye as minute glittering points, which the microscope resolves into large, bright triangular prisms or modified forms, sometimes feathery and sometimes having a resemblance to a "coffin-lid." (See Fig. 53.)

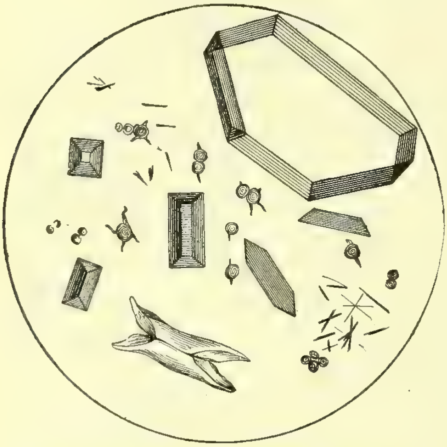
The existence of these mixed phosphates as a spontaneous deposit at the time of micturition usually indicates some serious bladder trouble, such as cystitis or paralysis or stone.

Incomplete evacuation of the bladder has favored the ammoniacal fermentation in the retained urine. If the condition persists, there is ground to fear that eventually a phosphatic concretion will form in the bladder and in the pelvis of the kidney.

Stellar calcium phosphate is sometimes deposited in urine which is alkaline from fixed alkali, and is rarely seen except in some general disorder of serious import. Commonly it occurs in arrow-heads or slender wedges, singly or gathered in star-like masses. (See Fig. 54.)

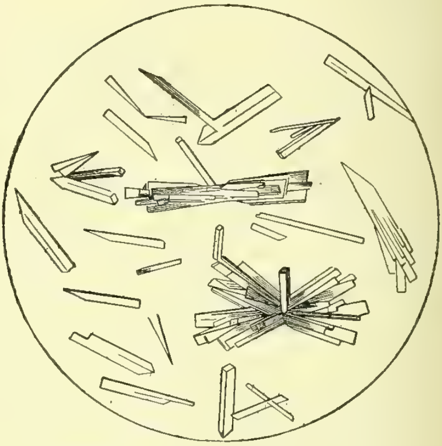
SULPHATES.—The sulphates of the alkaline bases ($K_2SO_4+Na_2SO_4$), which are eliminated by the urine to the extent of about 2 grammes, or 30 grains, daily, are derived partly from the diet and partly from the oxidation of the albuminoid principles of the tissues and fluids.

FIG. 53.



Triple Phosphate and Spheres of Ammonium Urate (Laache).

FIG. 54.



Stellar Calcium Phosphate (Laache).

Practical Import.—Although their amount is increased by fever and other wasting pathological conditions, the increase is not strictly proportional. The effect of diet is not easily calculated, nor can exact allowance be made for the uneven action of the eliminative process. On these accounts it is of no value to the clinician to determine the amount of the sulphates.

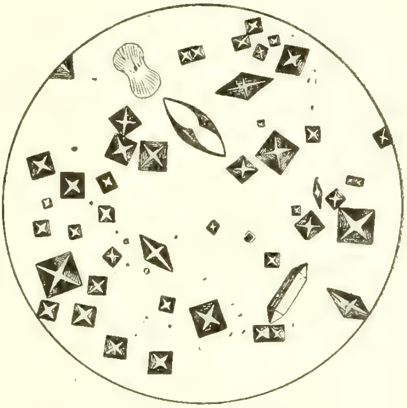
CHLORIDES.—Nearly 200 grains, or 8 to 12 grammes, of these salts (NaCl and KCl) are discharged daily by the urine. They are greater in amount than the sulphates and phosphates put together. In testing for them the sample is first acidulated with a few drops of nitric acid, so as to hold the phosphates in solution. Then, drop by drop, a strong solution of silver nitrate is added as long as white curds of silver chloride are precipitated. If the amount of chlorides is less than normal, then, instead of heavy curds being formed, the urine becomes milky or cloudy. A rough estimate can thus be made as to any marked deviations from the normal quantity. To make a volumetric determination put 10 c.c. of urine into a beaker and add 50 c.c. of water and a few drops of neutral potassium chromate. From a burette let fall by drops the standard solution of silver nitrate while stirring with a glass rod. As soon as the urine in the beaker becomes permanently orange-red throughout, read off the cubic centimetres used, subtracting 1 c.c. for excess of silver solution.

As the standard solution contained of $AgNO_3$ 29.06 gm. per 1000, equal to 10 gm. NaCl, so 1 c.c. = 0.01 NaCl in the 10 c.c. used, or 0.1 per cent. To get the percentage of chlorides, multiply the number of cubic centimetres noted by 10; to get the number of grains to the fluidounce, multiply the cubic centimetres by 45.57. Thus, if 13 c.c. were used, 12 c.c. would be counted. If 1 c.c. = 0.01 NaCl, then 12 = 0.12, or 1.2 per cent. While this method may be considered sufficiently accurate for clinical purposes, it fails in exactness when the urine is high-colored, albuminous, or putrid.

Practical Import.—It has been observed that in acute febrile diseases, such as pneumonia, pleurisy, and rheumatism, at the stage in which exudations are forming the chlorides are retained in the body, while they diminish in the urine. In cases of pneumonia with extensive exudation they may totally disappear. Their reappearance may be expected when resolution sets in and the fever declines. The missing quantity is made up by excess in convalescence.

Calcium Oxalate.—While oxalic acid is usually stated to be a constituent of the urine, the amount should not exceed what is called "a trace." Its combination with calcium is soluble to such an extremely small amount that anything more than a trace appears as a spontaneous deposit. Its naked-eye characters are not distinctive from those of mucus or epithelial débris. Its crystals are very minute, and call for the microscope to make out their form. It takes two different shapes, more commonly occurring as "octahedra," which appear as bright squares with diagonal cross-lines like envelopes, and sometimes, though rarely, showing as very small "dumb-bells," or forms resembling an hour-glass. (See Fig. 55.)

Fig. 55.



Calcium Oxalate (Laache).

Practical Import.—It is probable that a diet of subacid fruits and vegetables, like rhubarb or pie-plant, containing oxalates will furnish directly oxalic acid to the urine. As it is a laboratory product of oxidation-changes in fats, sugars, and starches, it is easy to see why impaired digestion or retarded metabolism may be the source. Although appearing sometimes without assignable cause, it usually attends conditions to be remedied by plain diet, temperance, and out-of-door life. It is frequently incidental to the gouty habit. The deposit may be transient, scanty, and unimportant, or it may be persistent and more abundant, and on these accounts serious, as indicating a disposition to the formation of a concretion of the kind known as the "mulberry" calculus.

Urea.—As it is the chief solid constituent of the urine, urea is also the most important physiologically as well as pathologically. Its chemical formula, $CO(NH_2)_2$, shows its nitrogenous character, and presents the view held as to its nature, a carbonyl diamide. The amount excreted daily is nearly 500 grains, or 40 grammes, equal to all the other solids put together; from which it will be seen what a conspicuous rôle is played by it as a compound representing the waste of the proteid or nitrogenized principles. Neutral in reaction, freely soluble in water and alcohol, though insoluble in ether, it is without color or odor, but has a bitter taste. In its chemical behavior, while it does not affect litmus, at times it is basic, and then again it may act in a compound as acids do. From concentrated solutions it can be crystallized slowly in quadratic prisms bevelled at the ends.

If the urine be evaporated in a water-bath to the consistency of syrup and nitric acid added to it, the crystals of urea nitrate should soon form in rhombic plates or hexagonal scales. Failure to precipitate crystals under these conditions (when the urine has been evaporated one-half) would indicate deficiency in the proportion of urea in the sample.

Urea is decomposed by boiling the urine; by the action of fuming nitric acid; by free chlorine or bromine or certain of their compounds, such as sodium hypochlorite and hypobromite:

$$N_2H_4CO + 3NaBrO = 3NaBr + 2H_2O + 2N + CO_2.$$

The above equation represents the reaction of urea with sodium hypobromite. As stated, it yields sodium bromide, water, free nitrogen, and carbon dioxide. By using with the sodium hypobromite an excess of alkali, such as sodium hydrate, as in Knop's fluid, the CO_2 is fixed in the solution as carbonate, and the only gas escaping is free nitrogen. The equation for this reaction should be,

$$N_2H_4CO + 3NaBrO + 2NaHO = 3NaBr + Na_2CO_3 + 4H_2O + N_2.$$

The most convenient methods for estimating the amount of urea depend upon the reaction given above. While most of them make use of the fact that the escaping nitrogen can be collected and measured, one of them, Fowler's, is based entirely upon the consequent loss of specific gravity. To get good results by *Fowler's method* requires the picnometer or a very accurate urinometer, with a scale so divided that fractions of a degree can be read.

Having noted the specific gravity of the urine, and also of the liquor sodæ chloratæ to be used, mix gradually 1 fluidounce of the former with 7 fluidounces of the latter. A brisk effervescence ensues, and may continue to some degree for two hours. At the end of that time take the specific gravity of the mixture. This number is subtracted from the mean obtained by adding to the specific gravity of the urine the product of seven times the specific gravity of the other fluid and dividing by eight. The remainder, multiplied by 0.77, gives the percentage of urea in the urine.

For example:

Sp. gr. of urine = 1018 × 1 volume = 1018.
" " liq. sodæ chl. = 1024 × 7 " = 7168.
1018 + 7168 = 8186.
8186 ÷ 8 = 1023, the mean sp. gr.
1023 − 1020 (sp. gr. of mixture after action) = 3.
3 + 0.77 = 2.31 per cent. of urea.

Owing to some cause, perhaps mainly the uncertain strength of the liq. sodæ chloratæ as commonly sold, this method often fails to give correct results. The experience of the writer is not favorable to it.

Hypobromite Method.—Different forms of apparatus have been devised to measure the free nitrogen evolved by the sodium hypobromite. The form usually employed is known as Russell and West's or as Apjohn's. The urine and the reagent are mixed, and the effervescing gas is delivered by a rubber tube at the top of a graduated cylinder open at the bottom and immersed in water, serving as a gasometer. This collecting cylinder may be graduated in cubic centimetres, and in such a case the determination will be made on the basis of 1 c.c. of nitrogen evolved for 0.0027 grammes of urea. Sometimes it is graduated to read percentage, on the principle that 5 c.c. of a 2 per cent. solution of urea will yield 37.1 c.c. of nitrogen. As solutions of sodium hypobromite are unstable, in order to ensure an accurate result it is best to prepare them freshly according to the formula of Knop, and if any great interval must elapse between successive determinations, it is best to keep the reagent in two parts, each of which is stable. Knop's solution is made by dissolving 100 grammes of sodium hydrate in 250 c.c. of water. This should be kept in a separate bottle, with a stopper of rubber or of glass coated with paraffin. When the fluid is to be used, measure out 15 c.c., and mix with it 1 c.c. of bromine. Care must be observed in pouring the bromine, as the vapor is highly irritating to the eyes and air-passages. The procedure is as follows: First immerse the graduated gasometer-tube upright until the water rises in it to the zero mark. Measure the 16 c.c. of Knop's fluid into the flask, and then by means of forceps or a string (to hold it upright, so as not to spill) put in a short test-tube containing 5 c.c. of urine. Close the flask tightly with a rubber stopper carrying a gas-tight rubber connection with the graduated gasometer. Now tip the flask so as to gradually pour out the urine into the surrounding hypobromite. By performing this slowly there is greater certainty that the CO_2 will be held fixed by the alkali, and that nitrogen only shall pass into the measuring vessel. After waiting ten minutes to complete the decomposition of urea, raise the gasometer so that the water within is on a level with that outside, and read off the cubic centimetres of nitrogen collected (or the percentage of urea if the tube be so graduated).

The computation is 0.0027 of urea in the 5 c.c. of urine used for each cubic centimetre of nitrogen. Thus, if the gasometer records 7 c.c. of nitrogen, then there were $7 \times 0.0027 = 0.0189$ grammes of urea in the 5 c.c. of urine. Multiplying by 20 to get the percentage, we have 0.0378 per cent.

To calculate grains to the fluidounce, multiply the percentage by 4.55. If the apparatus is stated to have been graduated at 70° F., reasonable accuracy in results can be obtained by taking the observation in an apartment at or near that temperature. The variation of temperature indoors would then affect the volume of gas so little as to be of small moment in clinical observations. In such cases differences of barometric pressure might be ignored without seriously affecting the calculation.[1]

Another nitrogen apparatus deserving of special commendation is that devised partly by W. D. Green and partly by Marshall. Still others are that of Lyon and that of Doremus. Space does not admit of detailed description of each of these, all of which are based on the same principle as that of Russell and West above described.

Liebig's Method.—Urea has the power of decomposing the mercuric nitrate and of forming a definite insoluble compound with the resulting mercuric oxide. This property is available for volumetric analysis, and, though less easy than the hypobromite process, it requires only the ordinary burettes and filtering apparatus, giving fairly good results. Several solutions must be made in advance: these are best made by a skilful pharmacist: 1st. A baryta solution, made by mixing 1 volume of cold saturated solution of barium nitrate with 2 volumes of cold saturated solution of barium hydrate. 2d. A saturated solution of sodium carbonate. 3d. A standard solution of mercuric nitrate, made according to official formula and preferably by a chemist. Process: To clear the way of interfering salts, first precipitate the phosphates, sulphates, and carbonates by mixing 20 c.c. of the baryta solution with 40 c.c. of urine. Filter, receiving 15 c.c. of the clear filtrate in a beaker. This represents 10 c.c. of the urine. Into this beaker, containing 15 c.c., now drop slowly from a burette the standard mercuric nitrate solution as long as fresh precipitate forms. The end of the reaction comes when the mercuric nitrate is in excess. This point is detected by taking a drop from the beaker and bringing it in contact with the sodium carbonate solution on a white plate. If it turns yellow, the process is complete. As the chlorides remaining in the urine have some effect on the reaction, allowance must be made by deducting 2 from the number of cubic centimetres used out of the burette. The remainder, multiplied by 0.010, gives in grammes the urea contained in the 10 c.c. of urine acted upon. This, multiplied by 10, gives parts per hundred.

Practical Import.—Urea, being freely soluble, never figures as a spontaneous urinary deposit. Morbid conditions causing increased tissue-waste will always run up the proportion of this product. In fevers and divers inflammations the amount is increased in the early or forming stage, and then declines with the febrile movement. In all acute diseases, as well as in phthisis, the

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[1] Some gas-measuring tubes are graduated to be read at a temperature of 32° F. or 0° C., and barometric pressure of 30 inches or 760 m.m.

Owing to the susceptibility of gas-volumes to variations of heat and pressure, to ensure perfect accuracy a correction must be made according to the following formula:

$$V' = \frac{V\ (b-w)}{760\ (1+0.00366T)}, \text{ in which}$$

V' = volume required; V = volume observed;
b = barometer in m.m; w = tension of aqueous vapor;
T = observed temperature centigrade.

rate of excretion rises and falls with the exacerbations of fever. In acute yellow atrophy of the liver at first it may be increased, but soon it declines notably, and in the end may disappear utterly. There is apt to be a marked lessening in the proportion when acute or chronic Bright's disease affects the eliminating powers of the kidney. Eventually this brings on the very dangerous symptoms of uræmia. When urine is retained in the bladder from diseases which interfere with complete evacuation, the ready conversion of urea, which is locally innocuous, into irritating ammonium carbonate causes it to figure as a pathological factor.

URIC ACID.—The chemical formula of uric acid, $C_5H_4N_4O_3$, shows its derivation from the nitrogenous principles of the body. While it resembles urea in containing nitrogen and in its origin, it is very unlike it in other respects. The average daily quantity excreted is only 10 grains or 0.7 grammes. Taking 18,000 parts of water for its solution, it may be considered as practically insoluble in that medium, though freely soluble in the alkalies and solutions of the alkaline carbonates. A trace of the free acid may be discovered in normal urine, but anything more than a mere trace will be precipitated, and then it has pathological significance. The 10 grains eliminated daily are not free, but combined with sodium and potassium as neutral urates soluble at ordinary temperatures. A dense urine kept long enough to pass into the acid fermentation will throw out the uric acid along with acid sodium urate and calcium oxalate. The acid can be separated from its basis artificially by adding 10 parts of hydrochloric acid to 100 of urine. After standing forty-eight hours minute brown crystals of uric acid will fall. To collect these, and thereby obtain an approximate estimate of the amount, the supernatant urine should be decanted, the sediment washed by stirring it with 30 parts of water, and then collected by throwing them with the water upon a weighed filter. After drying the filter with its sediment in a hot-air chamber, it can be weighed again, and then, allowing for the weight of the paper, the weight of the crystal can be ascertained.

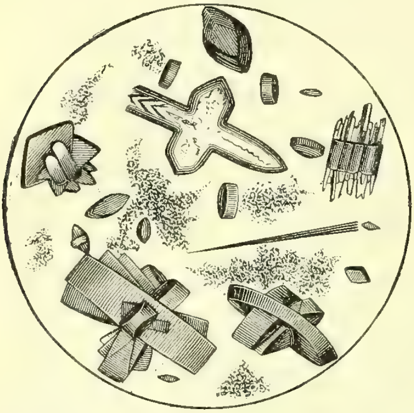
Uric acid free or in combination can be identified by the murexide reaction. To obtain this the suspected substance is treated in a watch-crystal or porcelain dish. After adding a few drops of strong nitric acid, enough to dissolve, a slow heat is applied to evaporate the solution to dryness. A yellow or reddish residue is obtained. This, touched with a drop of aqua ammonia or held over the open mouth of the bottle of ammonia, should turn to a bright crimson, purple, or violet (murexide).

If the crystals are examined under a microscope, they are found to be of a pointed oval form. As they fall they take up the coloring matter of the urine, which makes them red or brownish.

When the crystals fall spontaneously, they are larger, though still minute, and can be made out by the naked eye as the only brown specks of this size found in the urine. They are not unlike grains of red sand or ground cayenne pepper. Under the microscope they appear as small reddish lozenges, sometimes broken and single, sometimes united so as to form stars, rosettes, or sheafs. These are all modifications of the simple rhomb or "whetstone." (See Fig. 56.)

Practical Import.—Excess of uric acid may be one expression of the gouty diathesis, in which there is a tendency for the nitrogenous waste to take this shape in an undue proportion. Cases occur, not otherwise related to gout, in which the urine deposits free uric acid spontaneously. If this happens only when several hours have elapsed after micturition and as a consequence of the acid fermentation, it may be ignored safely. The same crystals, however, seen in a sample soon after micturition should awaken the suspicion that the sediment likewise falls in the bladder or in the kidney. If persistent these would aggregate into calculous masses. About 80 per cent. of all the urinary concretions are composed of uric acid along or mixed with urates. The solubility of these in alkaline fluids is the basis for medical treatment by the liberal exhibition of the alkaline bicarbonates or citrates.

FIG. 56.



Uric Acid and Mixed Urates (Funke).

MIXED URATES.—Under this title are included salts of uric acid with sodium, potassium, and perhaps other bases which in normal urine are soluble at ordinary temperatures. If, however, morning urine of ordinary acidity and density be kept in a cold room, its solvent powers are lessened, and it may become turbid, forming a surface film and throwing down these mixed urates as a loose pink powder. Even at common temperatures this sediment may occur if urine is very dense and of higher acidity than usual. In such cases the urates may not in themselves be in excess, but the urine, owing to hyperacidity, becomes a poorer solvent for them. These conditions are very frequent, and hence this deposit is a very familiar one: it is sometimes known as the lithate or the lateritious or the brick-dust deposit. It is not at all difficult to recognize, being the only deposit which clears up when the urine is heated. Again, it is dissolved when potassium hydrate is added to the urine. The same procedures would leave a phosphatic deposit unchanged, or even increase the turbidity. In case of doubt the murexide test (see Uric Acid) would act with the urates.

Microscopically, the urates are found to be amorphous granules with a tendency to form moss-like groups pinkish in color. To distinguish them from amorphous phosphates, a drop of potassium hydrate may be caused to flow under the cover. The urates will dissolve, but the phosphates are unaffected.

Practical Import.—By referring to the conditions producing them, it will be seen that before attaching much importance to this deposit it must be ascertained if the urine has been kept in a cold place. If not, then the deposit may be one evidence of excess of uric acid due to increased waste of nitrogenous tissue, such as occurs in fever. This may be transient, as from cold, or persistent, as in chronic diseases causing hectic fever. Sometimes it is the expression of a gouty habit, or it may be assignable to a free indulgence in meats and heavy liquors. The urine can be cleared up quite easily by making it alkaline, and sometimes by merely lowering its acidity. For this the usual remedy is potassium citrate in doses of half a drachm to a drachm, given in an effervescent draught. If the urates are persistently deposited while the urine is in the bladder, they tend to accrete about a nucleus, and thus gradually form a concretion.

If there is reason to suspect that a calculus has this composition, it may be advisable to try to dissolve it by a course of alkaline draughts maintained for long periods.

AMMONIUM URATE.—This compound of uric acid has some properties differing from those of the mixed urates referred to above. It will form as a deposit in urine made ammoniacal by putrescence, and then appears in company with the triple phosphate crystals. Under the microscope it is seen as dark-brownish spherules. Under this title some writers class a deposit made of irregular spherules with spiny projections. These have been called "hedgehog" crystals. Occasionally they look not unlike an acarus insect.

Practical Import.—The dark spherules are simply incidental to the ammoniacal fermentation. The spiny globes are sometimes seen in the dense, scanty, high-colored urine passed by children in febrile attacks. Concretions are very apt to be formed by them if the attacks are of frequent occurrence.

SUGAR.—The existence of even a trace of glucose in normal urine is, to say the least, doubtful. The generally accepted belief is that sugar is absent from healthy urine. Some of the urinary constituents, such as uric acid and kreatinin, can be made to exhibit reducing powers resembling those of glucose. In normal urine this power is not marked enough to appear distinctly with the usual reduction tests when properly made, whereas in true glycosuria it is shown to a pronounced degree. It is well to remember that high-colored, acid, and dense urines contain a relatively large amount of uric acid and kreatinin, and that with such samples additional care should be observed to avoid a fallacy. It is always advisable before testing for glucose to make sure of the absence of albumin.

Glucose as a Reducing Agent.—The most striking tests make use of the property possessed by glucose of reducing metallic salts to lower oxides, or even to the metallic state, when boiled with these salts and an excess of alkali. *Trommer's test* is made with half an inch of urine in the test-tube, to which is added an equal amount of potassium hydrate, liquor potassæ, and a few drops of a solution of copper sulphate. These are heated over a spirit lamp or a Bunsen's burner until boiling begins. A red or yellow precipitate of cuprous oxide denotes glucose. This is a crude and sometimes fallacious method of testing with copper sulphate. To obviate its defects, it is best to make the alkaline copper solution first and bring it to the boiling-point before adding the urine. But when the alkali and the copper sulphate are mixed, an objectionable precipitate of cupric hydrate forms. The change into an insoluble hydrate can be prevented by adding certain carbon compounds, such as the tartrates, glycerin, mannite, and glucose. Of these, however, glucose only has the power to abstract oxygen from the boiling copper solution, throwing down the red or yellow cuprous oxide.

The *glycerin cupric* test may be accurately applied by mixing in the tube an inch of potassium hydrate, a few drops of copper-sulphate solution, and a drop or two of glycerin. Having heated this mixture to boiling, about 10 drops of suspected urine should be added. After waiting a few seconds, if the yellow or red precipitate does not appear, the mixture must be brought to the boiling-point again and a few drops more of urine added. This process must be repeated until the yellow or red precipitate appears or until the total contents of the tube reaches 2 inches. The yellow or red precipitate denotes glucose. In practice it is very convenient to have the glycerin and copper ready mixed. This is done by dissolving 28 grains of copper sulphate in a fluidounce of pure glycerin. To make the test fluid, several drops of this are added to an inch of potassium hydrate. *Fehling's solution* differs from the preceding in using a tartrate as the medium for making a clear alkaline copper fluid. It may be made and contained in a single bottle, but in that shape does not keep well, depositing the red oxide of copper spontaneously. It is better to have its components in two separate bottles labelled A and B, of which equal parts are to be mixed when used. To make solution A, mix copper sulphate 34.64 grammes and water enough to make 500 c.c. For solution B, mix Rochelle salt 173 grammes, solution of sodium hydrate (sp. gr. 1.33) 100 c.c., and water enough to make 500 c.c. To make Fehling's solution mix equal parts of A and B.

Fehling's test is made by putting about one-half an inch of the above solution into a test-tube and diluting it with two inches of water. When heated to the boiling-point add a small amount of urine. If no red or yellow precipitate appears, heat to boiling again, and add another instalment of the urine short of an inch in amount. Heat to boiling again, and watch it as it cools; the slightest yellow or red turbidity would indicate glucose.

In all the above copper tests care should be taken that the test fluid should exceed the urine in volume, and that the contents of the tube should not be boiled, but merely heated to the point of boiling and then withdrawn from the flame.

Volumetric Estimation.—Having mixed in a porcelain capsule 10 c.c. of Fehling's solution and 40 c.c. of water, the mixture should be heated over wire gauze until boiling begins. While thus heating a burette may be charged with a mixture of 1 volume of urine to 9 of water. This diluted urine should be allowed to drop slowly from the burette into the gently boiling test fluid until the blue color of the copper solution totally disappears. Having noted the number of cubic centimetres required, if great accuracy is desired the whole process may be repeated with fresh materials, dropping the urine very slowly as the reaction approaches its end. In this way it is easy to determine the least amount required to decolorize the Fehling solution. The solution has been standardized, so that 10 c.c. of it will be decolorized by 0.05 grammes of glucose. If it be found that 7 c.c. of the dilute urine were needed, then, as the urine was diluted 1 part in 10, we read it 0.7 c.c. of urine = 0.05 grammes of glucose. Parts per hundred can be calculated by the ratio $0.7 : 0.05 :: 100 : x = 7.14$. To get grains to the fluidounce, the 7.14 must be multiplied by the factor 4.55.

Böttger's Bismuth Test.—As albumin may interfere with this test, owing to the sulphur it contains, it is desirable first to make sure that no albumin is present. If found it can be separated by making the urine slightly acid with acetic or nitric acid, boiling, and when cool filtering. About one inch of this urine (albumin-free) is put into a test-tube with one inch of liquor potassæ and a pinch of bismuth subnitrate. The mixture, being boiled for *several minutes*, will turn brown, and the white bismuth salt will turn gray or black if sugar is present. A convenient shape is given to the reagent by Nylander in the following solution, which contains both the alkali and the bismuth oxide: Take bismuth subnit. 2 parts, Rochelle salt 4 parts, and caustic soda (solution of 8 per cent.) 100 parts. Into a test-tube put two inches of urine and about one-quarter of an inch of Nylander's solution. After boiling a few minutes, change to a brown or black color would indicate glucose. The result with the bismuth test is not free from doubt until the fallacy due to sulphur compounds is eliminated. As they make a black precipitate with lead salts, which are not affected by glucose, litharge can be used to detect them. If when litharge is substituted for bismuth subnitrate in Böttger's test a brown or black color is produced, then sulphur compounds are present, and may cause a black precipitate, masking the test for glucose. Assurance can be made doubly sure by trying Fehling's test, which is free from liability to this fallacy.

Picric-Acid-and-Potash Test—About one inch of suspected urine is mixed in a test-tube with half an inch of the saturated solution of picric acid and half an inch of liquor potassæ. On boiling this yellow mixture for one minute a slight deepening of color may occur in normal urine, owing to reduction by uric acid and kreatinin; but change to a dark mahogany-red color would denote glucose.

Fermentation Test.—Reducing substances other than glucose, such as are derived from various drugs administered, are sometimes present and render the observer liable to a fallacy if he depends on the reduction tests only. Glucose is the only substance yet found in the urine which will pass into the alcoholic fermentation when brewer's yeast (Fleischmann's) is added to it and the mixture allowed to "work" in a warm place. After twenty-four hours the glucose will have disappeared, being resolved partly into carbon dioxide which escapes and partly into alcohol which remains. This breaking up of a dissolved solid into lighter, volatile parts occasions a loss of specific gravity in the solution proportionate to the amount of the solid involved. Not only is this an excellent test for the presence of glucose, but by the *Rob-*

erts method it is available for quantitative estimates. This differential-density process is simple, requiring an accurate urinometer, some brewer's yeast, and a bottle of urine.

The specific gravity is carefully taken and recorded; then about 4 ounces of the urine are thoroughly mixed with half a cake of Fleischmann's yeast and set aside in a warm place (the kitchen) for twenty-four hours. Fermentation will prove conclusively that the urine is saccharine. When the fermentation subsides the specific gravity is taken again and compared with the first observation. According to Roberts, each degree of density lost stands for 1 grain of glucose to the fluidounce of urine. If percentage be desired, the product must be multiplied by 0.219. For example: if the specific gravity before fermentation was 1040 and that taken afterward was 1020, then 1040 − 1020 = 20 grains of glucose to the fluidounce of urine. This 20 multiplied by 0.219 gives 4.38 per cent. Sometimes the test is performed by collecting the carbon-dioxide gas. To do this a test-tube must be filled with a mixture of urine and brewer's yeast, the thumb put over the mouth so that the tube may be inverted, and the opening immersed in a deep saucer containing the same mixture. The inverted tube, having been securely fixed, must be kept for twenty-four hours in a warm place. If glucose is present to an amount exceeding 0.1 per cent., some gas will collect at the top of the tube.

Practical Import.—The presence of sugar in the urine, or "glycosuria" as it is called, proceeds from conditions regarded as essentially pathological. In the majority of cases it is a sign of diabetes mellitus. In this disease the sugar is commonly abundant, sometimes reaching the large amount of 10 per cent. or 50 grains in the fluidounce: it persists for many months, and occasions the excretion of large quantities of urine, which may amount to 2 gallons daily, pale in color and of a mellow-apple odor. With the excess of water there is an increase in other natural constituents, such as urea. The total effect of these solids and the sugar is to raise the specific gravity above the normal point. At the same time, there is an obvious breaking down of the health: the patient grows emaciated, notwithstanding his voracious eating and drinking. The amount of sugar excreted and the cognate symptoms are measurably under the control of a dietetic regimen. By cutting off saccharine and amylaceous foods from the dietary, not only the proportion of sugar in the urine, but also the fluid volume, can be lessened.

It remains to be said that glycosuria is sometimes transient and slight. In some individuals, usually of stout build, it may appear as a consequence of excess in saccharine food. Temporarily, glucose or some other substance giving the same reduction reactions has been found after the administration of ether, chloroform, chloral, morphine, amyl nitrite, turpentine, salicylic acid, salol, benzoic acid, glycerin, camphor, carbolic acid, strychnine, arsenic, phosphorus, and carbon monoxide. Glycosuria may complicate various diseases of the brain and spinal cord, cirrhosis of the liver, cholera, phthisis, pneumonia, and asthma. In malarial regions a paroxysmal variety has been encountered.

ALBUMIN.—Of the several proteid compounds found at times in the urine, the two of greatest pathological import are serum-albumin and globulin. These two have certain differences, but they are both derived from the blood under like conditions and appear together in the urine. In practice it is not necessary to discriminate between them. The other albuminoids, such as mucin peptone and hemi-albumose, have, however, each a significance entirely different from that of albumin, though some of their reactions are similar. When albumin escapes into the urine it remains dissolved, as it does in the blood-serum, and can only be detected by reagents which change it to an insoluble compound called a coagulum.

Boiling Test.—Should the sample be cloudy, the portion to be tested must first be freed of suspended matter by filtration. This is easily and quickly done by resting the cone of filter-paper in the mouth of a test-tube. In a few minutes enough will be collected. When the turbidity is due to urates and apparatus for filtration is not at hand, gentle heat will serve to clear up the urine, and then by continuing the heat to the boiling-point the cloud of coagulated albumin will appear. The congeners serum-albumin and globulin are the only proteids that coagulate in acid fluids at 160° F. (70° C.), or even at 212° F. (100° C.), the boiling-point to which the heat is usually carried. The test is best made with about three inches of urine in the tube, and if the reaction be not acid it must be made so with one drop of acetic acid. Holding the tube aslant, the flame of the alcohol lamp or Bunsen's burner should be applied to the upper half only while the tube is slowly revolved. It is advisable to continue heating until boiling begins. If albumin be present, the heated half grows more or less cloudy as contrasted with the unchanged lower half. Two points must be emphasized: first, if the urine has its normal acid reaction, it is not necessary to add acetic acid; and, second, even when it is neutral or alkaline, only one drop of the acid should be used, lest the albumin should be converted into acid-albumin, which is not coagulated by heat. This test is available for making an estimate of the proportion of albumin. If the entire contents of the tube are boiled for a few minutes, and then set aside for twenty-four hours, the flakes of albumin will fall, so as to make a layer the volume of which can be stated as compared with the total depth of urine in the tube; thus, "the sample had $\frac{1}{10}$ or $\frac{1}{5}$ albuminous layer." It will be seen that this does not mean that the urine contains $\frac{1}{10}$ or $\frac{1}{5}$ part by weight of albumin.

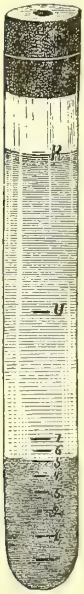
Nitric-Acid Test—Heller's Method.—If the urine is turbid, it must be made clear by pouring it through a cone of filter-paper set in the mouth of a test-tube. Having about one inch of clear urine, the tube should be inclined and nitric acid allowed to trickle down the glass, so as to form a bottom layer of about one-quarter of an inch in depth. After a few minutes, if appearances are doubtful, the tube should be held so that the light falls on it in such a way as to show up any haziness that may have formed. A more or less wide and distinct white belt at the line of contact of acid and urine indicates albumin. While this test used cold is not quite so sensitive as that by boiling, there are very few cases of serious albuminuria that cannot be detected by it. By keeping the acid and the urine separate, except at the line of contact, we ensure that at some point there will be just the amount of acid needed to coagulate the albumin. This method keeps the upper part of the urine unchanged, so as to be a standard for comparison.

There are cases where the reaction is so questionable as to make this standard of decided value. Occasionally a dense urine so treated will throw out a cloud of urates near the surface, but not at the line of contact. Hemi-albumose forms a white cloud with cold nitric acid. To make the conclusion positive, it is necessary to apply the boiling test in addition.

Picric-Acid Test.—The reagent is a saturated solution made by dissolving 6 grains of recrystallized picric acid in a fluidounce of hot water, and after standing for a time decanting the clear fluid. The urine must first be free from turbidity: if necessary for this, it may be dropped through a cone of filter-paper into the test-tube until about three inches collect. The picric acid is then permitted to flow down the side of the tube held standing to prevent the two fluids mixing. The yellow reagent remains on top, and if albumin is present a more or less cloudy zone will *immediately* form in the urine as far as the picric acid diffuses downward. If the upper part of the turbid zone is heated to the boiling-point, haziness due to albumin will increase, and, if the tube is set aside, will subside as a compact stratum resting on the unchanged column of urine below.

Besides albumin, the acid urates and several occasional constituents, such as mucin, peptone, and the alkaloids, will yield an opalescence to picric acid. But the albumin cloud is peculiar in that it persists after heating. This is a very delicate test; indeed, it sometimes reveals albumin in amounts so small as not to have significance for the practitioner. The same reaction is employed in estimating the weight of albumin by Esbach's albumimeter. This is a test-tube of strong glass graduated in the manner shown in Fig. 57. The test solution is prepared by dissolving 10 parts of picric acid and 20 of citric acid in 900 of boiling distilled water. After cooling, a sufficient quantity of water is added to make a total of 1000 parts. The object of the citric acid is to ensure that the liquid shall overcome any possible alkalinity in the urine. The graduated tube is filled with clear urine up to the mark *U*, and then the reagent up to *R*. It is then closed with a stopper, and the two liquids are thoroughly mixed in such a manner as to avoid shaking by slowly reversing the tube about ten times. Quick agitation might make air-bubbles that cause the precipitate to float. These must be removed with a pipette. After standing upright for twenty-four hours a dense and well-defined coagulum of albumin falls. The height of this sediment, read off on the etched scale, will indicate the weight of dried albumin in parts per thousand of urine (grammes per litre). While this process yields results which within a certain range are fairly accurate (an error of one-tenth to two-tenths of albumin), it is far more convenient than the tedious and difficult, though more accurate, method of

FIG. 57.



Esbach's Albuminometer.

separating the albumin by heat and acid and after filtration weighing the dried precipitate. Esbach's process will not give correct statements of amounts less than 0.5 parts per 1000. When the proportion of albumin is very high—that is, when the coagulum stands above 4 on the scale—it is best to dilute the urine with 1 or 2 volumes of water, and after testing multiply the result by 2 or 3, according to the degree of dilution.

In addition to the tests already given, which have the confidence of the profession and the sanction of much usage, there remain to be described several others less popular but of great sensitiveness:

Tanret's potassio-mercuric iodide reagent is composed of mercury bichloride 1.35 grains, potassium iodide 3.32 grains, acetic acid 20 c.c., distilled water enough to make 1000 c.c. By the contact method it shows a white belt with albumin, but also with other proteids whose presence may not be at all significant. The same objection can be made to the solutions of *sodium tungstate*, of *metaphosphoric acid*, and the more complex *acetic ferrocyanide* test. The last named is of extraordinary delicacy. It is applied by first making the urine decidedly acid with acetic acid, and then adding a few drops of recently prepared solution of potassium ferrocyanide. It precipitates albumin and also hemi-albumose.

A recently-introduced test is that by *trichloracetic acid*. It can be carried about the person as solid crystals, and put into the urine without previous solution, or used as a saturated solution after the "contact" method. In either case a white clot forms next to the reagent.

Practical Import.—Except in certain rare cases, such as the cyclical albuminuria of adolescents, albumin is an indication of a serious disturbance in the function of the kidney. It is generally conceded that at certain hours of the day a trace of albumin can sometimes be found in the urine of young men otherwise in apparent health. In small amounts it is often seen at certain stages of severe specific fevers and blood-poisonings, also during pregnancy and just after epileptic seizures. Poisoning by lead, arsenic, and some other metals may occasion it. In every such case the question arises, Can the albuminuria be regarded as a sign of Bright's disease of the kidneys? The answer will be affirmative if the symptom proves persistent and the layer produced by the boiling test should equal one-half of the column of urine in the tube. For proof positive one must examine the sediment with the microscope for tube-casts.

The general condition must be considered, and would be regarded as highly confirmatory if characterized by anæmia, cardiac hypertrophy, or dropsy. With these even a mere trace of albumin must be held to be of very grave import. Reactions of albumin with blood may be due simply to the hæmorrhage which may come from any part of the genito-urinary tract. When found with abundance of leucocytes, it may be due to the fluid of pus, and have no other significance.

PEPTONE AND HEMI-ALBUMOSE.—In testing for albumin, if a negative result follows upon using the combined tests of nitric acid and boiling, and yet a cloudiness is produced by cold acetic acid, there is reason to suspect the presence of peptone. This suspicion would be confirmed if response is given to the *biuret test.* In applying this, albumin, if present, must be removed by acidulation with nitric acid, boiling, and filtering. Then into a test-tube containing one inch of Fehling's solution of copper sulphate an equal quantity of the clear urine is poured gently down the inclined glass, so as to form a top layer. Any sample of urine will show a cloudy zone of phosphates at the line of contact, but if above this zone a rose-pink halo appears, then either peptone or hemi-albumose is present. If albumin is not removed in advance, it will mask the reaction, because it gives rise to a violet hue.

Practical Import.—When active suppuration is going on anywhere in the body and inflammatory effusions are breaking down, peptone and hemi-albumose are products. These seem to enter the systemic circulation, and in part at least to be eliminated by the kidney. They may thus help to prove the existence of concealed internal suppuration.

HÆMATURIA.—Blood imparts to urine the reaction of albumin contained in serum and a red or brown color due to the corpuscles. The change of color and the albumin reactions may be found in hæmoglobinuria, a condition in which the distinct corpuscles are not found, the color principle being diffused.

The characteristic feature of true hæmaturia is the red blood-corpuscle. These biconcave bodies will preserve their peculiar form for several days if the urine containing them is of ordinary density and acidity. In a very dense urine they lose their smooth outline and become crenated. In a dilute medium they swell up to a spherical shape and grow pale. In the ammoniacal urine which usually attends cystitis they are apt to shrivel and be deformed.

Practical Import—Hæmaturia is a symptom of hæmorrhage from some part of the genito-urinary tract. When the bleeding is at the kidney, the blood is usually well mixed with the urine, giving it a smoky-red appearance, and when the sediment falls bloody renal tube-casts can be found with the microscope. It denotes active local mischief, or may be symptomatic of severe fevers or neurotic or toxic or vicarious to menstruation and hæmorrhoids. Blood from the ureter is apt before evacuation to form clots which are moulded in that tube in the shape of curved cylinders, looking to the naked eye like small worms. They have been mistaken for parasitic entozoa. The microscope shows them to be a compact mass of red corpuscles. They may be due to local disease or injury, or incidental to the passing of a renal calculus. Blood from the bladder is usually abundant and gives to the urine a bright-red color, with shreddy clots visible to the naked eye. It is accompanied by vesical symptoms, such as pain in the suprapubic region and perineum, with frequent micturition and strangury. Blood from the urethra occurs in the course of gonorrhœa, and reveals its source by local symptoms and by escaping at the meatus in the intervals of micturition.

Heller's test for blood-pigment is made by adding liq. potassæ and boiling until flocculi of phosphates form. As they fall they carry with them the blood-pigment and become brown or red-yellow. It is an easy and satisfactory test.

HÆMOGLOBINURIA.—In certain "dissolved states of the blood" the coloring matter is set free from the disintegrated corpuscles and eliminated by the kidneys. It imparts to the urine a dark-brown color. The albumin reaction is obtained by all the tests for that substance. However, the coagulum formed is not white, but red or brownish. To distinguish this condition from hæmaturia the microscope is necessary.

In hæmaturia we not only have the color and the albumin reaction, but also the red corpuscles. The latter are not found in hæmoglobinuria. With the spectroscope the dark bands of reduced blood-pigment can be identified by the special means employed with that instrument. Almen's test by overlaying with urine a mixture of tincture of guaiac and a solution of hydrogen peroxide (or old ozonized oil of turpentine) gives a characteristic blue color.

Practical Import.—Hæmoglobinuria occurs in various blood diseases, microbic and otherwise, such as typhus, purpura, and pyæmia. Sometimes it is the result of the toxic action of hydrogen arsenide, phosphorus, carbolic acid, chloral, or potassium chlorate. Certain individuals suffer from a periodic form, often attributable to cold or malaria, and sometimes of doubtful origin.

BILE.—In conditions causing jaundice we can always find bile-pigment in the urine, but the biliary acids are seldom present in amounts great enough to give the lake-colored reaction with the well-known Pettenkofer's test by cane-sugar and sulphuric acid.

Oliver's test by peptone solution is very sensitive, but gives results so uncertain as not to merit detailed description in a practical study of the urine as brief as this is required to be. On the other hand, the bile-pigment can be detected in the urine of icterus even earlier than it will show itself on the conjunctiva. When notably present it gives tints varying from bright sulphur-yellow to olive-green.

Gmelin's test is very sensitive and easily performed. A few drops of the suspected urine are poured in a white plate, and near them a small amount of yellow nitric acid (containing lower oxides of nitrogen). Having caused the two fluids to touch edges, bile-pigments will change at the line of contact into modified pigments. There will be a play of colors in regular order—green, blue, violet, red, and yellow. Green and red dominate, and will persist after the others fade. The same test can be applied in a tube by overlaying the nitric acid with the biliary urine.

Practical Import.—A trace of bile found will help to diagnose hepatic troubles when the icteroid hue elsewhere is doubtful.

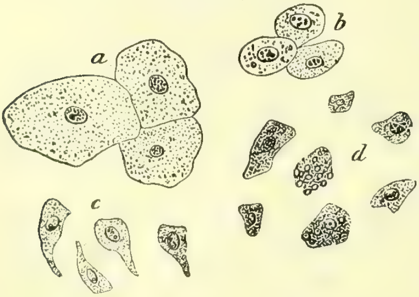
PYURIA.—It has been stated above that sometimes the albuminuria may be due to pus, the fluid of which is albuminous. The distinctive elements of pus are the numerous leucocytes. These under the microscope can be recognized by their resemblance to the white blood-cells. They are spherical, granular, and opaque, but on the addition of acetic acid lose their opacity and show at the centre one, two, or three nuclei. One cannot be sure from the form whether the leucocyte is derived from mucus or from pus. With the former comparatively few are to be found, with the latter a great number. Mucus can be further distinguished from the pus from the fact that the proteid mucin will not become hazy with cold nitric acid, while the albumin of liquor puris coagulates like serum-albumin. Again, if the suspected sediment is separated by decanting the upper part of the urine, and then into the deposit a piece of caustic potassa is stirred, if the deposit is pus it becomes tough and gelatinous; if mucus, thin and flaky.

Practical Import.—In pyuria the albumin reaction raises the question as to whether in addition to pus there is serum-albumin of renal origin. We are helped to a conclusion by the fact that the albumin in pyuria is usually scanty, and a large amount would therefore be considered as over and above that due to pus. If tube-casts are found with the microscope, then renal mischief can be assumed. A sudden irruption of pus would most likely be due to the evacuation of an abscess into the genito-urinary passages. Persistent pyuria points to chronic catarrhal inflammation, the site of which can be determined by local symptoms.

Chyluria.—Chyle is rarely found in the urine. At first sight of a sample containing it one would suppose that milk had been added to it. It may happen that the amount of chyle present is so large that the fat-particles rise like the cream on milk, and the fibrin of the chyle may form a spontaneous clot resembling *blanc-mange*. As the chyle contains serum-albumin, it would respond to the tests for that substance. To make out the fatty character of the molecular basis, a portion of the urine should be agitated with ether, which dissolves the fat-particles and melts them together as a surface layer, leaving the urine clear beneath. The microscopic character is much like that of milk —*i. e.* it contains myriads of small bright round particles which dissolve in ether.

Practical Import.—This symptom generally appears in those who have lived in the tropics, where it is not very uncommon. It denotes a lymphatic connection with the urinary passages, and not infrequently is associated pathologically with the presence of the filaria sanguinis hominis.

Fig. 58.



Epithelium from the Urine; *a*, *b*, epithelium from the bladder, from the pelvis of the kidney; *c*, candate epithelium (pelvis of the kidney?); *d*, renal epithelium, partly changed into fat (Vierordt).

Epithelium.—Ordinarily the urine is clear, but even in health it occasionally shows a faint cloud called the nubecula, which the microscope reveals to be made of epithelial débris. In some persons a small amount of the waste material of cells from the mucous lining of the bladder and other parts of the urinary tract may occur, and have no significance. A large amount with mucus, or still more with pus, would indicate catarrh of some portion of the urinary tract. Practically, the main point to be determined is as to whether the cells are from the kidney or not.

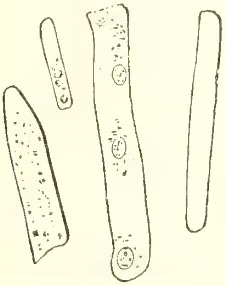
Renal epithelium is spherical, granular, and nucleated, with an indistinct cell-wall. The coexistence of casts of the uriniferous tubes would corroborate their testimony as to the existence of renal desquamation. Cells from extra-renal parts are distinct, nucleated, and flattened, being oval, spindle-shaped, cylindrical, or tessellated, according to site. Cylindrical or caudate cells may be derived from the pelvis of the kidney, from the prostate gland, from Cowper's gland, from the urethra, or from some parts of the bladder. Bladder epithelium is usually flat and irregularly oval; sometimes desquamation occurs in patches of cells joined at their edges. In the urine of women large translucent flat cells from the vagina are nearly always present.

Tube-casts.—As a result of structural mischief in the kidney, there are formed in the tubules cylindrical casts of coagulable material, which is sometimes fibrin, sometimes mucoid matter, and sometimes the plastic substance resulting from the disintegration of the cellular lining. Individually they are too small to be seen by the naked eye, but in the amount usually collected they appear as a light gray sediment, or perhaps as a cloud at or near the bottom of the glass vessel. Under the microscope they are seen to be minute cylinders, glassy or opaque, and granular or displaying cells. They can be classified accordingly as epithelial, hyaline, granular, fatty, and those made of blood-disks.

Epithelial casts have opaque spherical renal cells imbedded in some plastic matrix. By the number of these one can judge of the activity of the desquamative process in cases of nephritis. They are usually found in acute nephritis. (See Fig. 63.)

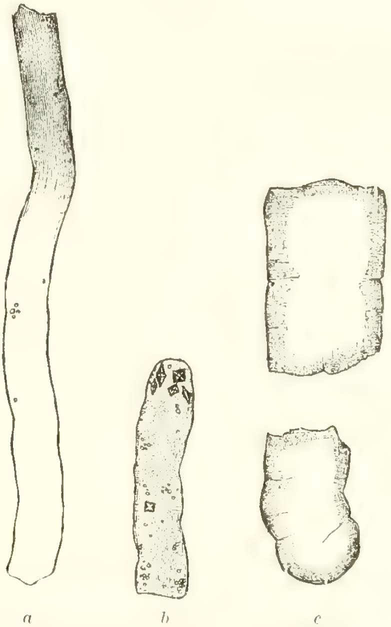
Hyaline casts are always transparent, and sometimes require skilful arrangement of light to show them at all. They can be grouped in two sub-varieties, in one of which, the mucous, would be placed those that are soft and of delicate outline; in the other, the waxy, those that are well defined and brittle.

Fig. 59.



Hyaline Casts (narrow and tolerably broad ones).

Fig. 60.

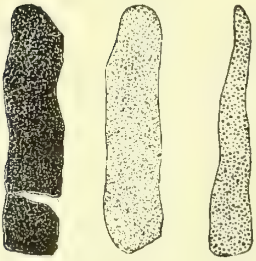


a and *c*, Waxy Casts (Jaksch); *b*, a cast containing crystals of oxalate of lime.

The mucous casts alone are sometimes found without any other sign of nephritis, and hence must be regarded as not always of serious import. The waxy casts, on the contrary, are never found but when the kidneys are diseased. (See Figs. 59 and 60.)

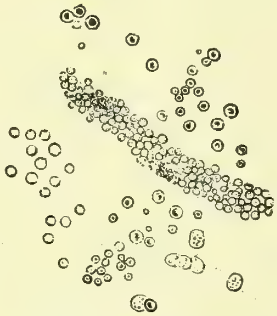
Granular casts (Fig. 61), as the name indicates, are composed of or contain opaque granules which have a yellowish hue. The material may be mucoid or waxy, or such material as is produced by cellular débris.

FIG. 61.



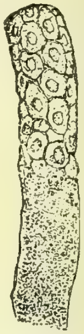
Granular Casts (Jaksch).

FIG. 62.



Red Blood-corpuscles, partly as "rings," and Casts of Red Blood-corpuscles (Eichhorst).

FIG. 63.



Epithelial Cast (Jaksch).

Fatty casts are such as have fat-particles in the matrix, with or without the other bodies mentioned above. If numerous, they are regarded as evidence of fatty change in the kidney.

Blood-casts (Fig. 62) are reddish and opaque; they are literally minute clots of blood which have taken shape from the tubules into which the effusion occurs. The corpuscles may be so packed as to be pressed out of their biconcave shape and appear as reddish circles.

Practical Import.—It has been stated under previous sections that if albuminuria or hæmaturia or an epithelial deposit be of renal origin, careful search of several portions of the deposit with the microscope will most likely find tube-casts. It occasionally happens in cases of Bright's disease that the albuminuria will disappear, and still the casts can be found in the urine. Hence much importance is attached to them in renal diagnosis. As regards the significance of particular varieties, it must be noted that if the mucous cast alone is present it does not prove nephritis, but any of the other varieties would do so.

It often happens that several varieties occur in the same sample: this probably denotes that the lesion is at different stages in different parts of the organ.

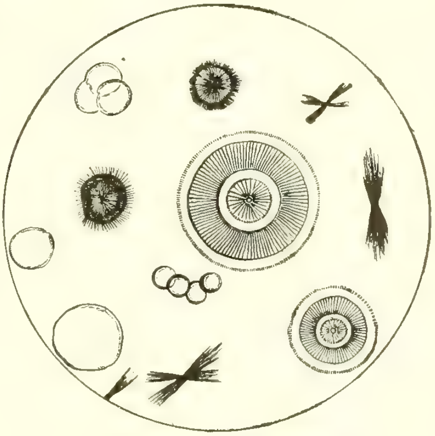
CYSTIN.—This substance contains sulphur, the composition being expressed by the formula $C_6A_{12}N_2S_2O_4$. One product of its decomposition is the gas hydrogen sulphide; hence a test for it is to boil the suspected material with a solution of lead oxide in sodium hydrate. If cystin be present, it will form a black precipitate of lead sulphide. As it is very sparingly soluble in water, any considerable amount in the urine would not remain in solution, but be deposited. The deposit is usually abundant, light, and to the naked eye resembles amorphous urates. Unlike urates, it is not dissolved by heat, though it is soluble in ammonia and also in the vegetable acids. When a drop of the ammonia solution is exposed uncovered on a glass slide, it deposits crystals which the microscope shows to have the form of hexagonal tablets.

The extensive use of iodoform for surgical dressings has been the source of a fallacy. The crystals of iodoform, accidentally mixed with the urine and viewed by the microscope, will present hexagonal tablets not unlike those of cystin. The chemical reaction is wholly different, and the pronounced odor of iodoform should at once excite suspicion.

Practical Import—In very small amounts cystin may be a constituent of healthy urine. Certain individuals and families, from causes not ascertained, have persistent excess with deposits. These evidently form the very rare cystin calculus.

Leucin and Tyrosin.—These two substances are considered together, because they are by-products of the same processes of digestion, and when from disease certain biliary matters appear in the urine the other can be found also. Tyrosin is recognized by its turning red when boiled with Millon's reagent of mercurous nitrate: when another portion is carefully warmed with sulphuric acid, and then treated with a drop of ferric chloride, it yields a violet color. In the urine tyrosin may be in solution or it may be thrown down spontaneously as a greenish-yellow deposit. The microscope will resolve this deposit into bundles of yellow acicular needles in radiating stars, crosses, or sheaves.

Fig. 64.



Leucin and Tyrosin (Laache).

Leucin, being more soluble, is less apt to form a spontaneous deposit, but if a few drops of the suspected urine are allowed to evaporate by exposure on a glass slide, both leucin and tyrosin will appear in the residuum. Under the microscope leucin is recognized as greenish-yellow globes with concentric marking or radiating spines. If the deposit is touched with a drop of nitric acid, cautiously evaporated to dryness, and then moistened with sodium hydrate, the leucin residue will turn yellow or brown.

Practical Import.—These two bodies are found with icterus in certain maladies when the liver is seriously involved, as in acute yellow atrophy of the liver, phosphorus-poisoning, typhoid and yellow fever.

Spermatozoa.—These bodies, if present in considerable number in the urine, form a whitish cloud. When taken up with the pipette the sperm detaches as a thready, drop-like viscid mucus. When only a few are present, they impart no marked naked-eye property, and in looking for them with the microscope unless a proper oblique light is used they may escape observation. In the urine they lose at once their vibratile motion, and yet for days retain their structural characters, the small transparent oval body or head with the very attenuated cilium, the whole being only $\frac{1}{600}$ inch long.

Practical Import.—Before drawing conclusions as to their significance, it must be ascertained if the sample containing them be not the first micturition after coitus. If not, they may be the washing out of the remains of a nocturnal emission of semen. Their only important relation is as an indication of spermatorrhœa, the escape of sperm independent of the sexual act or of its physiological equivalent, the nocturnal emission occurring in a continent person.

MICRO-ORGANISMS.—As it is a fluid containing more or less organic matter in solution, the urine is a fertile medium for the development of microscopic vegetation. The spores or germs of these minute plants come from the containing vessels or from the dust that floats in the air.

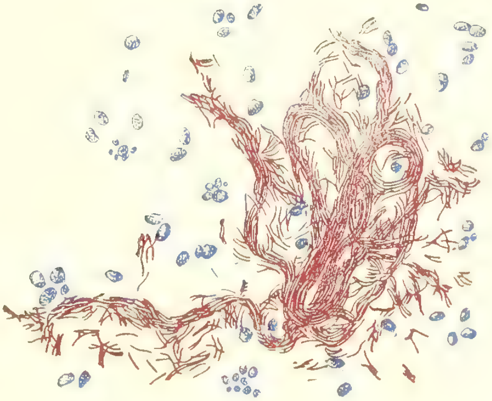
The common moulds, such as penicilium, appear in a few days on stale urine. They are seen microscopically as minute jointed threads matted together in a mycelium. Saccharine urine furnishes the soil for the growth of the yeast fungus, saccharomyces cerevisiæ, the spores of which may be derived from the floating dust of the air. It may be of value as corroborating other evidence of the presence of sugar. The latter plant is recognized as oval cells, with granular contents and nuclei multiplying mainly by buds, but sometimes by spore-bearing stems. Even before discharge the sarcinæ of the bladder will reproduce in the urine, and be the cause of obscure vesical symptoms. Their microscopic structure is peculiar from the cubic form of the little masses made by the reproduction of the more minute round particles. The bacteria of putrefaction, the micrococcus ureæ, vibriones, and other similar organisms will flourish not only in the urine outside, but even before micturition. They are identified as extremely minute rods or granules, single or threaded, still or vibratory.

Practical Import of the Bacteria.—As yet, the only assured significance of bacteria in the urine depends upon the fact of their causing decomposition of the urea while still in the bladder. If the ammoniacal products are detained in the bladder, they are very apt to cause cystitis. It is of the greatest importance to guard against the introduction of their germs by means of unsterilized instruments, such as catheters and sounds. It is possible for them to get access to the urine in the bladder from the purulent discharges of a deep-seated gonorrhœa or gleet. In paralysis of the bladder they appear to have the power of spontaneous entrance. In that event the harm they may do must be obviated, as far as possible, by frequent and thorough evacuations of the bladder and washings with antiseptic fluids.

When the specific pathogenic bacteria are looked for, it must be with high-power immersion lenses and substage condensers after drying and staining the residue by the approved methods of bacteriology.

It must be noted, on the authority of Lustgarten and Mannaberg, that the urine in passing through the urethra of healthy subjects may wash out micro-organisms that colonize there. Among these is mentioned a diplococcus resembling the gonococcus of gonorrhœa in all respects save that it is not found in pus-corpuscles, a large streptococcus, and even a bacillus which neither by form nor by staining can be distinguished from the tubercle bacillus. The last-named is usually seen singly, whereas the bacilli coming from ulcerating uro-genital tuberculosis are generally in groups or crowds considerable in number, like those of a pure culture (Fig. 65). Inoculation experiments would serve to distinguish them from the non-tubercular bacilli. The coexistence of hectic fever and wasting with pyuria and masses of these bacilli in the sediment would prove highly confirmatory of their tubercular origin.

Fig. 65.



Pure Culture of Tubercle Bacilli in the Urine in Tuberculosis of the Genito-urinary Apparatus. (Zeiss's homogeneous immersion one-twelfth eye-piece No. 4; drawn with a camera lucida; magnified about 1100. Vierordt.)

When the pathogenic bacteria are made out in the sediment unmistakably by form and number, they point to the specific associated disease—the tubercle bacilli to miliary tuberculosis, the erysipelas cocci to erysipelatous nephritis, the pus micrococci to pyæmia or endocarditis.

The hooklets of echinococcus may be found in the urine, denoting the presence of hydatid cysts somewhere in or about the urinary apparatus. Other parasites occasionally seen in the urine of persons who have lived in the tropics are distoma hæmatobium, strongylus gigas, and filaria sanguinis.

DISEASES OF THE KIDNEYS.

BY FRANCIS DELAFIELD.

THE KIDNEYS are deeply seated in the lumbar region, lying one on each side of the vertebral column, at the back part of the abdominal cavity and behind the peritoneum. They are on a level with the last dorsal and the two or three upper lumbar vertebræ, the right kidney being a little lower than the left.

The kidneys measure about 4 inches in length, 2½ inches in breadth, and 1¼ inches or more in thickness. The left is usually longer and narrower than the right. The weight of each kidney is usually stated to be about 4½ ounces in the male, and somewhat less in the female.

It is difficult to determine changes in the size and position of the kidney, unless the enlargement is considerable or the displacement marked.

In order to map out the kidneys by percussion the patient must lie on his belly; auscultatory percussion is preferable to simple percussion. We mark a point 3½ inches outward from the spines of the vertebræ at the level of the eleventh rib on either side, then measure downward from this point 4 inches and mark a second point, and then join these two points by a space 2½ inches wide. Over the central portions of the area thus included we may expect to get a change in the percussion note due to the presence of the kidney. It must be admitted, however, that it is very difficult to reach certain results by percussion alone.

Palpation is used with the patient lying on his back. Any considerable enlargement or displacement of the kidney can be made out in this way. Enlarged kidneys, as a rule, are not movable by palpation nor by the movements of the diaphragm. The colon is as a rule pressed forward by the enlarged kidney.

CONGENITAL MALFORMATIONS.

These are of more importance to the surgeon than to the physician.

Absence of one kidney, the horseshoe kidney, the malpositions of the kidney are of frequent occurrence, and must be constantly reckoned with in operations on these organs.

THE MOVABLE KIDNEY.

It not infrequently happens that one, or sometimes both, kidneys cease to be imbedded in the fat of the lumbar region and become capable of a liberty of motion which increases as time goes on. The kidney continues to be fastened by its large blood-vessels, and pushes the peritoneum in front of it.

It is believed that such a movable condition of the kidney may be either congenital or acquired. It occurs more frequently in women, in women who have borne children, among the laboring classes, and in persons between the ages of twenty-five and forty years. The right kidney is the one most frequently affected, next the left kidney, least often both kidneys.

Symptoms.—We can only be certain of the existence of a movable kidney when we are able to distinctly feel it. It feels like a solid tumor, not as hard as the spleen, of the shape of the kidney, movable, easily escaping from the hand which tries to grasp it, easily pushed back into its natural position. In order to make out such a kidney it is usually necessary to put the patient into different positions, so as to displace the organ ; it is often necessary to examine on several different days before we can be sure of it.

Such a movable condition of the kidney may never cause any discomfort, and the patient may remain entirely unconscious of its existence. In other cases, however, the abnormal position of the kidney may give rise to a variety of symptoms.

Perhaps the most ordinary state of affairs is for the patient either to suffer from severe pain in the back or to discover that he has an abdominal tumor.

The pain in the back is referred to the side on which the kidney is displaced. It is a severe pain, made worse by the erect position. The real cause of the pain is often overlooked for a long time.

The discovery of an abdominal tumor naturally fills the patient with alarm, and so may give rise to a variety of nervous phenomena for which the displacement of the kidney can hardly be held responsible.

Less frequently, the patients complain of a variety of symptoms. There are feelings of dragging, of pressure, or of weight in the abdomen. There may be nausea, vomiting, and intestinal pain. Intense radiating pains may occur toward the epigastrium, the sacral and lumbar region, the intercostal spaces, the shoulders, along the ureter, into the testicle or the labia majora.

It is said that there may be attacks of localized peritonitis around the inflamed kidney, with chills, fever, intense pain, and hardness and tenderness of the abdomen.

The pelvis of the displaced kidney may become inflamed or may contain calculi.

By the bending of the ureter there may be produced, first, a temporary, and later a permanent, hydronephrosis.

Treatment is called for only in those patients in whom the displacement of the kidney gives well-marked symptoms. The simplest plan is to keep the patients in bed on their backs for a month, and then to let them go about with a binder and pad so contrived as to keep the kidney in place.

If the patient cannot be relieved in this way, a surgical operation becomes necessary. The offending kidney may be entirely removed or it may be fastened in its proper place by sutures.

THE URINE.

The examination of the urine gives us information concerning the condition of the kidneys, of their pelves and ureters, and of the bladder. Its composition is also affected by disturbances of digestion and by the presence of sugar.

The quantity of the urine in a healthy adult is about fifty ounces, changing at different times with the quantity of fluids ingested and the degree of perspiration.

A complete occlusion of the pelves of the kidneys or of their ureters leads to complete suppression of urine—a condition which is certainly fatal, but can yet be borne for a number of days without symptoms. Sooner or later, however, prostration, delirium, stupor, the typhoid state, and death follow.

Severe injuries and surgical operations, especially those on the urethra and bladder, may be followed by a suppression of urine which is often fatal. It is probable that this suppression is due to an acute congestion of the kidneys.

Acute and chronic congestion of the kidney and acute nephritis regularly diminish the quantity of urine. In chronic nephritis the quantity of the urine varies much at different times, being sometimes diminished, sometimes increased.

The urine is increased in quantity in chronic nephritis with a slow growth of interstitial connective tissue, or when there is waxy degeneration of the capillary tufts of the glomeruli, or with diabetes mellitus and insipidus.

The specific gravity of the urine is a matter of the greatest importance. To determine[1] it we must make a number of examinations, and allow for the quantity of fluid ingested by the patient. With these precautions a continued low specific gravity signifies diabetes insipidus, a chronic productive nephritis, or waxy degeneration of the blood-vessels of the kidney.

The urine may contain blood. This occurs most frequently with acute nephritis and with exacerbations of chronic nephritis. It is also found with tubercular nephritis and with malignant growths. It is a frequent symptom of pyelitis and of calculi in the pelvis of the kidney. Profuse bleeding has been seen from the atrophied kidneys of chronic nephritis.

The urine may contain albumin. Serum-albumin and serum-globulin are normal constituents of the serum of the blood. Their presence in the urine means that the serum of the blood has exuded from the blood-vessels into the kidney tubules. Such an exudation of serum from the blood-vessels in any part of the body is an inflammatory exudation or a dropsy.

In inflammation we are still ignorant of the precise reasons for the escape of the blood from the vessels.

As to the mechanism of dropsy, a number of explanations have been offered none of which are very satisfactory. We only know that disturbances of the circulation which produce venous congestion are also likely to produce dropsy; that persons in whom the relative quantity of the solid constituents of the blood is diminished are liable to have dropsy; and that it also occurs in ways for which we are unable to account.

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[1] For fuller details as to methods to be employed the reader is referred to the section upon Practical Urinary Examination.

One can sometimes observe for years persons who habitually have dropsy of the ankles, and are yet apparently perfectly well.

Scattered through medical literature are reports of cases of general dropsy coming on suddenly, lasting for a short time, and terminating in recovery, for which no cause could be discovered. Traube, indeed, thinks that such dropsies are due to a disturbance of the functions of the skin caused by exposure to the weather, but this explanation certainly will not answer for all the cases. In some[1] the dropsy was preceded by a well-marked febrile movement. I only know of one case of this kind which terminated fatally. It is reported by Wernicke.[2] The patient, a girl twenty-two years old, died apparently from the dropsy, and the autopsy showed no lesion to account for the symptom.

I owe to Dr. W. F. Milroy of Omaha[3] the account of a family in which dropsy of the legs was a congenital peculiarity. The family history was as follows: The maternal grandfather during most of his life had dropsy of the legs, and died of pneumonia, aged seventy-eight. He had four daughters. In three of these dropsy of the legs was congenital and continued through life; in one it was developed later in life. One of these daughters died in childbirth, aged thirty-eight; the three others are still alive, aged sixty-six to eighty-one years. Of the grandchildren, one, a man aged thirty-one, shows the same condition. He is a large, healthy, powerful man, who has œdema of both feet and legs extending nearly to the knees. The legs are twice their normal size and pit deeply on pressure. The œdema is increased by standing, but never entirely disappears.

While there are many tests for albumin in the urine, altogether the most satisfactory one is that by heat and nitric acid. The urine is filtered, a long test-tube is nearly filled with it, a few drops of acetic acid are added, and the upper portion of the column of urine is boiled. We then look at the test-tube with a good light and against a black background. If there is an opacity, we add a few drops of nitric acid to see whether the precipitate is dissolved or not. Other tests are superfluous.

Peptone and propeptone are said to be the products of gastric and pancreatic digestion of albuminous substances, and are also produced in the process of transformation of tissues and of inflammatory effusions. They are found in the urine with or without serum-albumin. Their presence in the urine is an evidence of disturbances of digestion, and not of any disease of the kidney.

Peptone is precipitated by metaphosphoric acid, acidulated brine, picric acid, potassio-mercuric chloride. The precipitate is dissolved by heat.

Propeptone is precipitated by nitric acid, metaphosphoric acid, acidulated brine, picric acid, potassio-mercuric chloride, potassium ferrocyanide. The precipitate is dissolved by heat.

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[1] Taylor, *Medical Times and Gazette*, 1871.

[2] *Deutsch. Arch. für klin. Med.*, vi. 622.

[3] *N. Y. Med. Journal*, Nov. 5, 1892.

Casts are cylindrical bodies formed in the kidney tubules by the coagulation of substances contained in the serum exuded from the blood-vessels. They are composed of a transparent, homogeneous matter, with which may be mixed renal epithelium, pus-cells, red blood-cells, and the granular matter, fat, and nuclei derived from degenerated renal epithelium. So we find hyaline, epithelial, pus, blood, nucleated, granular, and fatty casts. These casts may remain in the kidney tubules or may come away with the urine. The number of casts which we find in the urine is usually, but not always, an indication of the number formed in the kidneys. As albumin and casts are both the evidences of exudation from the blood-vessels, we expect to find them both in those forms of nephritis in which the quantity of exudation is considerable. In isolated cases, however, although the albumin is present in large quantities, the casts may be few in number.

Tubercle bacilli are found in the urine with tubercular inflammation of the bladder, ureters, pelves of the kidney, and of the kidney itself.

ALBUMIN IN THE URINE WITHOUT DISEASE OF THE KIDNEY.

Although albumin in the urine as a rule belongs to a nephritis with exudation or to congestion of the kidneys, yet it is also frequently met with in persons who have no disease of these organs. We find it in the following groups of cases:

1. PAROXYSMAL OR CYCLIC ALBUMINURIA.—This occurs most frequently in young males who are anæmic, poorly nourished, have headaches, neuralgic pains, bodily and mental languor, hysteria, and disturbances of the functions of the stomach, liver, and intestines. The albumin is absent at night and present at certain fixed hours every day. It is increased by the erect posture, by food, and by mental and bodily exertion. It disappears while the patient is in bed. The quantity of albumin may be considerable, but there are few casts.

In these patients the exudation of serum from the vessels of the kidneys seems to be of dropsical character, like the œdema of the legs seen in anæmic patients.

The treatment of these patients is like that of patients suffering from simple anæmia—the regulation of the diet and mode of life and the administration of iron. With such treatment most of the patients seem to recover.

2. DIETETIC ALBUMINURIA.—This occurs at all ages. It may follow the ingestion of only certain kinds of food—cheese, pastry, and eggs; or of any kind of food; or of any food which is not properly digested; or of food followed by exercise. The quantity of albumin is small, and there are few or no casts.

If this form of albuminuria is only temporary, it is not a serious condition, but if the disposition to it persists, the patients are to be regarded with suspicion. They do not necessarily develop kidney disease, but they are apt to die from some serious organic disease.

Treatment is to be directed to the digestive system.

3. Albuminuria after Exertion.—The exertion must be severe and prolonged. The quantity of albumin may be considerable, and casts are also present. In these cases, again, the point of importance is how easily the albuminuria is produced by exercise, and how often its production is repeated. It seems probable that the over-exertion causes acute congestion of the kidneys, and that a repetition of attacks of acute congestion would be followed by a nephritis.

4. Simple Persistent Albuminuria.—These patients may for years have small quantities of albumin in their urine nearly every day, without any other symptoms. Such persons are always to be looked upon with suspicion. They may do well for a number of years, but sooner or later disease of the kidneys, of the heart, or of the arteries is apt to be developed.

This last set of cases is of especial importance to life-insurance companies. Now that the urine of all persons who apply for life insurance is examined, it is found that, excluding the cases of kidney disease and the cases of temporary albuminuria, there are many persons who have a little albumin in the urine nearly every day. Such persons, even if they do not turn out to have kidney disease, are not "good risks."

Acute Congestion of the Kidneys.

Definition.—A temporary congestion of the blood-vessels of the kidney, which may be so severe as to produce exudation of serum and escape of red blood-cells.

Etiology.—Acute congestion is caused by the ingestion of certain poisons, by extirpation of one of the kidneys, by severe injuries inflicted on any part of the body, by surgical operations, especially those on the bladder and urethra, and by over-exertion.

Morbid Anatomy.—The only change in the kidneys is the engorgement of the blood-vessels; the tissue of the kidney remains unaltered.

Symptoms.—Acute congestion may occur in kidneys previously normal or in those already diseased. The urine is diminished in quantity or entirely suppressed. The urine which is passed may contain blood, albumin, and casts; its specific gravity is unchanged.

After the ingestion of a poison, such as cantharides, the diminution in the quantity of the urine, and the presence of albumin, casts, and blood, are the only decided symptoms.

If the congestion follows the removal of the other kidney, the urine is scanty or suppressed, albumin and casts are present, there is a good deal of prostration. These symptoms may last for a few days, and then disappear, or they may continue; the patients become more and more feeble, and pass into the typhoid state with mild delirium.

If the congestion follows injuries or surgical operations, the urine is diminished or suppressed. The patients pass into a condition approaching collapse, from which they may recover or in which they may die.

If it follows over-exertion, the urine is not much diminished in quantity, but albumin and casts are present. Single attacks of this kind are not dangerous, but a repetition of them seems likely to cause nephritis.

Treatment.—In all cases of acute congestion the patients are to be kept in bed, on a fluid diet, with the bowels freely opened. The surface of the body should be brought into a condition of perspiration, and this is to be effected by the hot pack or the hot-air bath.

Chronic Congestion of the Kidneys.

Definition.—A long-continued engorgement of the blood-vessels of the kidney, which is liable to be followed by chronic nephritis.

Etiology.—Chronic congestion of the kidneys can be produced by any cause which interferes with the circulation of the blood in such a way as to cause venous congestion of the viscera. The most common of such causes are—chronic inflammation of the aorta or mitral valves, dilatation of the heart, aneurisms of the arch of the aorta, pulmonary emphysema, and large accumulations of fluid in the pleural cavities.

Morbid Anatomy.—The congested kidneys remain of their natural size or become somewhat larger. They are heavy in proportion to their size. They are of hard consistence, of dark-red color, or the cortex is pink or white, while the pyramids are dark red. The capsules are not adherent, the surfaces are smooth. The epithelium of the cortex tubes remains unchanged, or the cells are flattened, or they are swollen and separated from each other. Of the glomeruli, some remain unchanged, but a considerable number are large, their capillaries dilated and with thickened walls, and the cells which cover the capillaries swollen. There are no changes in the stroma, except in some cases an increase in the size of the normal areas of subcapsular connective tissue. The arteries are, as a rule, unchanged, but sometimes the small arteries of the cortex are dilated and their walls thickened. The veins in the pyramids are congested and sometimes dilated. Much less frequently the capillary veins of the cortex are also dilated.

Symptoms.—The symptoms are for the most part those of the primary disease—the endocarditis, the dilatation of the heart, the aneurism, the emphysema, or the pleurisy. The principal effect of the congestion of the kidneys is to diminish the quantity of urine. Although the kidneys are always congested, they are much more so at some times than at others; so the quantity of urine is sometimes normal, sometimes diminished, sometimes suppressed. Under the effect of drugs the quantity of urine may be for a time increased. The specific gravity varies from 1.020 to 1.025, but it may for a time be down to 1.010 or up to 1.035. The quantity of urea to the ounce is increased rather than diminished. Albumin and casts are not a prominent feature. They may be entirely absent, but more frequently, when the heart-action is at its worst, they are present in moderate quantities. The effect of the congestion of the kidneys on their functions is, therefore, simply to diminish the quantity of the urine; its quality remains good, and the exudation from the blood-vessels is inconsiderable.

The most serious feature of chronic congestion is its liability to be succeeded by chronic nephritis.

Treatment is directed to the primary lesion which causes the congestion.

ACUTE DEGENERATION OF THE KIDNEYS.

Definition.—An acute change in the kidneys, characterized by degeneration or death of the epithelial cells of the tubules, to which may be added an exudation from the blood-vessels.

SYNONYMS.—Acute Bright's disease; Parenchymatous nephritis; Parenchymatous degeneration.

Etiology.—Acute degeneration of the kidneys seems to be always due to the introduction of poisons into the body. The poisons are inorganic, such as arsenic, mercury, and phosphorus, or those which accompany the infectious inflammations and diseases. With any infectious disease sufficiently severe to cause death it is the rule to find the kidneys in the condition of acute degeneration. It must, however, be remembered that some of the infectious diseases, such as scarlatina and diphtheria, may cause either acute degeneration or acute nephritis—two conditions which are entirely distinct from each other.

Morbid Anatomy.—The kidneys are more or less enlarged; the cortical portion is somewhat thickened, usually pale, occasionally congested; the surfaces are smooth, the capsules are not adherent. The changes in the renal epithelial cells are—a simple swelling of the cell-body, causing it to look larger and more opaque and to take on irregular shapes; an infiltration of the cell-bodies with granules of albuminoid matter and fat; a death of the cells, which may take the form of coagulation-necrosis or of a disintegration of the cell-bodies; a desquamation of the dead cells; a formation of hyaline masses in the cells; a growth of new cells to take the place of the dead epithelium. All these changes are most marked in the convoluted tubes. It may happen that the degeneration in the cells is developed with such intensity that the blood-vessels are irritated and there are added congestion and exudation of serum.

Symptoms.—If such an acute degeneration follows the ingestion of arsenic, mercury, or one of the other inorganic poisons, the urine is diminished in quantity or suppressed; it contains albumin, casts, and blood; its specific gravity is unchanged. At first the symptoms of the poison predominate, but if these subside and the degeneration of the kidneys continues, the patients become feeble, pass into the typhoid condition, and die, apparently from the kidney lesion.

The acute degeneration which accompanies the infectious diseases, such as scarlatina, diphtheria, typhoid fever, etc., is for the most part of mild type, not dangerous to life, and gives no symptoms except the presence of a little albumin and a few casts in the urine.

In some of these diseases, however, especially with yellow fever and acute yellow atrophy of the liver, the degeneration is of intense type, with death of a large part of the renal epithelium and exudation from the blood-vessels. In such cases the urine is scanty and contains albumin, casts, and blood. The patients are very ill and often die, but it is hard to tell how many of their symptoms are due to the kidney lesions.

Treatment.—So far as the degeneration of the epithelium is concerned, we know of no treatment which is likely to affect it favorably. But if exudative inflammation, with scanty and highly albuminous urine, is added to the degeneration, there is an indication to employ the methods of treatment appropriate to acute exudative nephritis.

CHRONIC DEGENERATION OF THE KIDNEYS.

Definition.—A chronic degeneration of the epithelial cells of the convoluted tubes.

SYNONYMS.—Chronic Bright's disease; Chronic parenchymatous nephritis; Fatty kidney.

Etiology.—The same mechanical interferences with the circulation which cause chronic congestion may also cause chronic degeneration. It is also caused by some of the chronic diseases, by chronic alcoholism, and by vicious modes of life. Like acute degeneration, it is a secondary process.

Morbid Anatomy.—The kidneys are, as a rule, increased in size, weighing together from sixteen to twenty ounces. Sometimes, however, they are of medium size or even quite small. Their surfaces are smooth, the cortex is pink, white, or yellow, the pyramids are red. The epithelium of the cortex tubes is swollen, coarsely granular, or infiltrated with fat. The glomeruli are unchanged, unless the degeneration be due to venous congestion. There are no changes in the stroma. The arteries are normal or their walls are a little thickened.

Symptoms.—The quantity of the urine varies at different times in the same case, and also in different cases; it may be abundant, scanty, or suppressed. Its specific gravity is not diminished, nor is the proportion of urea to the ounce of urine decreased. Albumin and casts in moderate quantities are often present. The patients become anæmic, lose flesh and strength, and pass into the typhoid condition with delirium and stupor. Dropsy and disturbances of the circulation are not developed, unless the degeneration is caused by disease of the heart or of the arteries.

Chronic degeneration may be followed by chronic nephritis.

Treatment.—The best that we can do for these patients is to stop all vicious habits, to regulate the diet and mode of life, and to relieve the disturbances of circulation; but it must be admitted that when the disease has begun it is apt to progress.

ACUTE EXUDATIVE NEPHRITIS.

Definition.—Acute inflammation of the kidneys, characterized by congestion, exudation of the blood-plasma, emigration of white blood-cells, diapedesis of red blood-cells, to which may be added changes in the renal epithelium and in the glomeruli.

SYNONYMS.—Acute Bright's disease; Parenchymatous nephritis; Tubal nephritis; Desquamative nephritis; Catarrhal nephritis; Croupous nephritis; Glomerulo-nephritis.

Etiology.—Acute exudative nephritis is frequently a primary disease, either occurring after exposure to cold or without discoverable cause. It complicates scarlatina, measles, diphtheria, typhoid fever, acute general tuberculosis, pneumonia, peritonitis, dysentery, erysipelas, diabetes, and many other of the infectious diseases and inflammations. It is one of the forms of nephritis which complicate the puerperal condition.

Morbid Anatomy.—In a nephritis of this type we should expect that the inflammatory products, the serum, white and red blood-cells, and coagulable matter from the blood-plasma would collect in the Malpighian bodies and tubes or infiltrate the stroma between the tubes, and that, of the inflammatory products in the tubes and Malpighian bodies, a part would be discharged with the urine and a part found in the kidney after death. We should also expect that the quantity of inflammatory products would be in proportion to the severity of the inflammation, and that an excessive number of pus-cells would belong to the especially severe forms of the disease. Still further, it is evident that with the milder examples of nephritis, with but little exudation, no inflammatory products might be found in the kidney after death, all having been discharged into the urine during life.

As a matter of fact, the kidneys do present just such changes. In the mild cases we find no decided lesions in the kidney after death. In the most severe cases the kidneys are increased in size, their surfaces are smooth, the cortical portion is thick and white or white mottled with red, or the entire kidney is intensely congested. If the stroma be infiltrated with serum, the kidney is succulent and wet; if the number of pus-cells be very great, there will be little whitish foci in the cortex.

In such kidneys we find the evidences of exudative inflammation in the tubes, the stroma, and the glomeruli, all the changes being most marked in the cortical portion of the kidney.

The epithelium of the convoluted tubes is often simply flattened. As this same appearance is also found in the chronic congestion of heart disease, it seems probable that this change of the shape of the cells is merely due to the inflammatory congestion.

In other cases not only is the epithelium flattened, but there is also a real dilatation of the cortical tubes. This dilatation does not involve groups of tubes, but all the cortical tubes uniformly. In other cases the epithelium of the convoluted tubes is swollen, opaque, degenerated, and detached from the tubes. The tubes, whether with flattened epithelium or dilated, may be empty. More frequently, however, they contain coagulated matter in the form of irregular masses and of hyaline cylinders. The irregular masses are found principally in the convoluted tubes; they seem to be formed by a coagulation of substances contained in the exuded blood-plasma, and are not to be confounded with the hyaline globules so often found in normal convoluted tubes. The cylinders are more numerous in the straight tubes, but are also found in the convoluted tubes.

They also are evidently formed of matter coagulated from the exuded blood-plasma, and are identical with the casts found in the urine. The tubes may also contain red and white blood-cells.

In the cases in which there is an excessive emigration of white blood-cells we find these cells in the tubes, in the stroma, or distending the capillary veins. This excessive emigration is not necessarily attended with exudation of the blood-serum, and so the urine of these patients may contain no albumin. The white blood-cells are not usually found equally diffused through the kidneys, but are collected in foci in the cortex. These foci may be very minute or may attain a considerable size.

In the glomeruli we find changes which, at first sight, seem peculiar, but are really similar to the changes which we find in arteries and capillaries in many inflamed tissues. The cavities of the capsules may contain coagulated matter and white and red blood-cells, just as do the tubes. The capsular epithelium may be swollen, sometimes so much as to resemble the tubular epithelium.

The most noticeable change, however, is in the capillary tufts of the glomeruli. These capillaries are normally covered on their outer surfaces by flat, nucleated cells, so that the tuft is not made up of naked capillaries, but each separate capillary throughout its entire length is covered over with these cells. There are also flat cells which line the inner surfaces of the capillaries, although not uniformly, as is the case in capillaries in other parts of the body. Still, in spite of the presence of all these cells, the outlines of the walls of the capillaries are fairly distinct.

In exudative nephritis the swelling and growth of cells on and in the capillaries change the appearance of the glomeruli. They are larger and more opaque; the outlines of the main divisions of the tuft are visible, but those of the individual capillaries are lost. It is difficult to tell how much these changes in the glomeruli interfere with the passage of the blood through their capillaries. In most cases of exudative nephritis the patients recover and the glomeruli return to their natural condition. The walls of the smaller branches of the renal artery may be thickened by a swelling of the muscular coat.

Symptoms.—In the milder cases, except for the changes in the urine, there is little to call attention to the kidneys, and as a matter of fact these mild cases of nephritis are not only often overlooked, but many physicians are not aware of the frequency with which they occur. The patients are hardly confined to bed, they have a little headache, perhaps some aching in the back and limbs; there may be a little nausea; they have the feeling of general malaise; they often think that they have taken cold. These indefinite symptoms last for one or two weeks, disappear, and the patient is well again. If the urine be not examined, it is not known that the patient has been suffering from a mild nephritis. If it be examined, it is found that the quantity is somewhat diminished, the specific gravity is not lowered, an appreciable quantity of albumin is present with hyaline, granular, and epithelial casts, sometimes with red and white blood-cells. If the number of blood-cells be sufficient to color the urine, the patient's attention is attracted by the change in color; the diminished quantity he is apt not to notice. These changes in the urine last for four or five weeks, and then disappear.

In the more severe cases the changes in the urine are more decided. It is diminished in quantity, or even suppressed; its specific gravity is normal or high; the quantity of albumin is very large; the casts are numerous—hyaline, granular, containing white or red blood-cells or epithelium; there are also free white and red blood-cells and epithelial cells from the kidney and the bladder. As a rule, the quantity of albumin and the number of casts are in proportion to the severity of the nephritis, but this is not always the case. Large quantities of albumin, numerous casts, and many white and red blood-cells may be found in the urine of kidneys which, after death, show no structural changes; while, on the other hand, small quantities of albumin and a few hyaline casts are compatible with a severe nephritis. Still further, the number of casts found in the urine during life is not always in proportion to the number of casts and quantity of coagulated matter found in the corresponding kidneys after death.

In addition to the changes in the quantity and composition of the urine, the patients present constitutional symptoms which vary, in the different cases, as to their number and their severity. A febrile movement, with more or less prostration; stupor, headache, sleeplessness, restlessness, muscular twitchings, and general convulsions; dyspnœa, loss of appetite, nausea, and vomiting; a pulse of high tension with exaggerated heart-action or hypertrophy of the left ventricle, dropsy, and anæmia,—these may be called the characteristic symptoms of acute exudative nephritis. Of these symptoms a certain number—the fever, the prostration, the loss of appetite and nausea, the anæmia, the diminution in the quantity of urine, the albumin and casts in the urine—are such as would naturally accompany an acute inflammation of the kidney, and very often they are the only symptoms which do accompany it.

With this same kidney lesion, however, a certain number of patients will present, besides the symptoms just mentioned, the additional features of cerebral symptoms, change in the heart and circulation, and dropsy. We indeed might think that the cerebral symptoms and the dropsy were due to the diminished excretion of urine; but when we know that these conditions occur, as they often do, in patients who are passing large quantities of urine of good specific gravity, and when they are absent, as they often are, in patients who are hardly passing any urine at all, it is evident that these symptoms are not directly due to the quantity of urine, but constitute a separate, complicating set of symptoms which may be present or absent in any given case of the disease.

What the complicating lesion is which produces thêse symptoms we do not fully know; but the changes in the action of the heart and the dropsy naturally direct our attention to the arteries and capillaries, with the expectation of finding in them some morbid condition which will hinder the passage of the blood through them. Whether the morbid condition be of an inflammatory nature, or whether it be only a spasmodic contraction, we are as yet ignorant.

The cases of exudative nephritis, with an excessive production of pus-cells, have a somewhat different clinical history. Such a nephritis occurs both in children and adults ; it may be primary or complicate scarlatina, diphtheria, or measles.

The invasion is sudden, with a high temperature and marked prostration. Restlessness, delirium, headache, and stupor are soon developed and continue throughout the disease. The patients lose flesh and strength and pass into the typhoid state. Dropsy is slight or absent altogether. The urine is not so much diminished in quantity as one would expect ; its specific gravity is not changed ; albumin, casts, and red and white blood-cells are present in considerable quantities, but not always early in the disease, and they may even be absent altogether.

Although this form of exudative nephritis is not of common occurrence, the unusual character of the symptoms and the great mortality are reasons for calling special attention to it. It differs from the ordinary form of exudative nephritis in that it behaves like an infectious inflammation, and that, although the emigration of white blood-cells is large, the exudation of serum may be small, and so the urine may show but little change.

The very mild cases of acute exudative nephritis run their course within four or five weeks ; the patients are not seriously ill ; they recover completely.

The more marked cases as a rule exhibit symptoms for as long as four weeks, and the albumin and casts in the urine continue for weeks afterward. The inflammation of the kidney, therefore, although of acute type, is apt to require as long as eight weeks to run its course.

Prognosis.—The patients who for three or four weeks have only the fever, prostration, loss of appetite, and nausea, anæmia, dropsy, and changes in the urine, as a rule recover completely, and are not at any time in real danger.

The development of the cerebral symptoms—the stupor, headache, sleeplessness, restlessness, muscular twitchings, and general convulsions—always causes anxiety, but yet even of these patients the larger number get entirely well.

The cases with an excessive production of pus-cells differ in their clinical history from all the other examples of acute exudative nephritis, and as a rule prove fatal.

Treatment.—We have to treat an acute exudative inflammation of which the natural tendency is to run its course in four weeks and to terminate in recovery. We have also to treat the symptoms of this nephritis—the scanty urine, dropsy, vomiting, anæmia, and cerebral symptoms. We have to treat these conditions more frequently in children than in adults, and very often as complicating an infectious disease.

It is to be remembered that, although the quantity of urine voided is small, its quality is good, for it contains a fair proportion of excrementitious solids to the ounce of fluid ; that convulsions and coma belong to the early days of a nephritis, while prolonged anuria is accompanied rather with the typhoid state; that the excretion of urine must naturally relieve the congestion of the kidney, so that it is better for the kidney to perform its functions than to be at rest; that so long as the congestion of the kidneys persists the quantity of the urine will be diminished; that a considerable diminution in the quantity of urine, continued for one or two weeks, is often perfectly well borne.

It is not, therefore, necessary to pay attention to the diminished quantity of urine, and to try to make the skin or the intestines do the work of the kidneys; it is wiser, by relieving the congestion of these organs, to enable them to do their own work, knowing that the moment they begin to do this their congestion will be still further diminished, and that, although the nephritis still continues, the excretion of urine may then be sufficient.

Fortunately, we have at our command means by which the congestion of the kidney can be diminished—not completely, it is true, but partly; not for any length of time, but for short periods.

These means are—causing the blood to collect at the surface of the body by the application of heat to the entire skin; the use of dry cups or wet cups or heat over the lumbar region; the empirical use of calomel or sulphate of magnesium in small doses, repeated at short intervals until the bowels begin to move; the use of such drugs as will diminish the increased arterial tension. While the nephritis is still active, however, we do not expect that by the use of any means the urine will be brought up to its full normal quantity, but only that a sufficient quantity will be passed to ensure the safety of the patient.

The febrile movement in an acute nephritis requires no treatment.

The prostration, loss of appetite, nausea, and vomiting only call for rest in bed and a fluid diet.

The anæmia ought unquestionably to be prevented or relieved, but while the nephritis is still active I know of no way in which this can certainly be done. When convalescence is established, then the anæmia readily improves with the ordinary methods of treatment.

The cerebral symptoms are those to which most attention has been directed. There can be no question that they accompany a contraction of the arteries with increased tension and labored action of the heart. No matter what views one may entertain as to the cause of this change in the circulation, I believe that treatment is best directed to the arteries themselves, rather than to the uncertain cause of their contraction. Fortunately, we possess drugs which act promptly and efficiently for this purpose. Of these drugs, the most suitable are aconite, nitro-glycerin, chloral hydrate, and opium, preferably given in small doses and at regular intervals before the cerebral symptoms are marked, but in large doses hypodermically or by the rectum to stop a severe attack.

It is wise to watch the condition of the heart and arteries, and as soon as the condition of increased arterial tension is developed, not to wait for the manifestation of the cerebral symptoms, but to try to relieve it at once.

The way in which we manage the patients, therefore, is as follows: They are put to bed or kept in the house until the nephritis has run its course. They are put on a fluid diet, preferably milk, and the skin of the entire body is cleansed every day. For many cases no other treatment is necessary.

If vomiting be troublesome, it can usually be controlled by adding oxalate of cerium and bicarbonate of sodium to the milk. For the restlessness and sleeplessness chloral hydrate, the bromides, or opium may be employed.

If the nephritis be evidently of severe type, we apply heat to the surface of the body and give either $\frac{1}{4}$ to 1 grain of calomel or 1 drachm of sulphate of magnesium every hour, until the patient has taken six doses or the bowels begin to move. This is followed by 1 or 2 drops of tincture of aconite every hour.

Throughout the disease we watch the pulse, and as soon as it shows any increased tension give chloral hydrate in doses of from 2 to 5 grains every three hours.

If severe headache, muscular twitchings, or general convulsions occur, we give chloral hydrate in doses of from 5 to 20 grains by the rectum, or nitroglycerin in doses of from $\frac{1}{200}$ to $\frac{1}{50}$ of a grain hypodermically, or morphine in doses of from $\frac{1}{10}$ to $\frac{1}{6}$ of a grain hypodermically.

As the nephritis subsides the milk is gradually replaced by solid food, and iron and oxygen are given.

ACUTE PRODUCTIVE (OR DIFFUSE) NEPHRITIS.

Definition.—An acute inflammation of the kidneys, characterized by exudation from the blood-vessels, a growth of new connective tissue in the stroma, and changes in the glomeruli.

SYNONYMS.—Acute Bright's disease; Parenchymatous nephritis; Croupous nephritis; Glomerulo-nephritis.

Etiology.—This is the most serious and important of the forms of acute nephritis, for the reason that its lesions are from the first of a permanent character, so that it is apt to persist and to be followed by chronic nephritis.

It is one of the forms of scarlatinal nephritis, it occurs early and late in the course of diphtheria, it is the most important variety of the nephritis of pregnancy, and it is especially frequent as a primary nephritis, with or without a history of exposure to cold. It is more frequent in children and young adults than in older persons.

Morbid Anatomy.—The kidneys are large, smooth at first, roughened in cases of longer standing. The cortical portion is thickened, white, or mottled with yellow or red, or congested; the pyramids are red.

In these kidneys we find the same lesions as have been described as belonging to exudative nephritis, but with two additional changes—changes which are found in the earliest stages of the inflammation, and which give the characteristic stamp to the lesion: first, a growth of connective tissue in the stroma; second, a growth of the capsule-cells of the Malpighian bodies.

Both these changes do not involve the whole kidney, but symmetrical strips or wedges in the cortex which follow the line of the arteries. These wedges are small or large, few or numerous, regular or irregular, in the different kidneys. But in every wedge we find the same general characters: one or more arteries of which the walls are thickened; the Malpighian bodies belonging to this artery showing an enormous growth of capsule-cells, with compression of the tufts; and running parallel to these arteries a growth of connective-tissue cells and of basement-substance in the stroma. Between the wedges we find at first only the changes of exudative nephritis; later, a growth of diffuse connective tissue. Sometimes we find these wedges small, symmetrical, and at considerable distances from each other; more frequently they are much closer together, sometimes even becoming continuous.

If the nephritis be of acute type and recent, the new tissue between the tubes consists largely of cells; if the nephritis be of subacute type and longer duration, the tissue is denser and has more basement-substance. Where the growth of new tissue is abundant the tubes become small and atrophied.

In each wedge is one or more of the arteries which run up into the cortex and give off the little branches ending in glomeruli. The walls of these arteries are thickened.

The glomeruli belonging to these arteries become the seat of changes of a permanent character. There is the same growth of the cells covering the vessels, and of the cells within them, as in exudative nephritis, reaching even a greater development. In addition, there is a growth of the cells lining the capsules to such a degree as to form a mass of cells compressing the tuft. The tuft apparently never returns again to its natural condition, but as time goes on the vessels are obliterated, the capsule-cells are changed into connective tissue, and the glomeruli are finally transformed into little balls of fibrous tissue.

Symptoms.—Of the patients who suffer from this form of nephritis, a certain number behave as if they had a simple exudative nephritis. They have a febrile movement, with more or less prostration; stupor, headache, sleeplessness, restlessness, muscular twitchings, and general convulsions; dyspnœa, loss of appetite, nausea, and vomiting; a pulse of high tension with exaggerated heart action, or hypertrophy of the left ventricle, dropsy, and anæmia. The urine is scanty or suppressed, it is colored by blood, and contains much albumin and many casts. They are, however, much more likely to die within a few days or a few weeks than if the nephritis were of simple exudative type.

The patient, instead of dying, may at the end of about four weeks apparently recover, and all the constitutional symptoms disappear. It will, however, be found that the albumin and casts persist in the urine; after weeks or months renal symptoms again appear, and the case takes on the characters of a chronic nephritis.

Of much more frequent occurrence are the cases in which the invasion of the symptoms is less violent. The urine is only moderately diminished in quantity, it often contains no blood, there is a good deal of albumin, but the casts are not numerous.

Some of the patients complain at first of nothing but the dropsy of the legs. They continue to eat well, feel well, and attend to their work. In others, anæmia, headache, sleeplessness, dyspnœa, nausea, vomiting, or diarrhœa are present in addition to the dropsy. The patients do not feel well or disposed to exert themselves, and yet many of them are not confined to bed.

Beginning in this way, some of the patients get steadily worse. The dropsy involves more and more of the body, they lose flesh and strength, the anæmia becomes more profound, they may have attacks of contraction of the arteries with general convulsions, or may pass into the condition of alternating delirium and stupor with a feeble pulse. Many months, however, elapse before the fatal termination of the disease is reached.

In other cases the first attack lasts but a few weeks, and the patients, except for the changes in their urine, seem to be well. After weeks or months, however, another attack with the same symptoms comes on; then, after another interval of apparent recovery, another attack; and so the case progresses until finally an attack comes which proves fatal. It is only the first attack in these patients which can be regarded as an example of an acute nephritis. As the disease goes on it must be looked upon as a chronic nephritis with exacerbations, and the specific gravity of the urine will steadily be lowered.

Prognosis.—An acute productive nephritis usually terminates fatally. It may either destroy life in its character of an acute inflammation, or by so damaging the kidneys that chronic inflammation is sure to follow. But yet the prognosis is not altogether unfavorable; in many patients life and fair health can be prolonged for many years; in a few entire recovery seems to take place.

Treatment.—In those cases in which the disease behaves like an acute exudative nephritis the indications for treatment are the same as in the latter disease, although the results are not as satisfactory. In these patients it is of great importance to insist on prolonged residence in a proper climate after the the subsidence of the acute symptoms.

In the cases which begin more subacutely, with dropsy, anæmia, and disturbances of the stomach, the nephritis does not require active treatment; the application of heat to the surface of the body and the use of calomel, sulphate of magnesium, or aconite is not necessary. Rest in bed at first, and residence in a proper climate later, have more effect on the nephritis than has anything else. Too much stress cannot be laid upon the importance of the selection of a suitable climate—generally a warm, dry, equable climate, where the patient can lead an out-of-door life.

The patient should at first be kept on milk alone, but this must not be continued for too long a time; after a few weeks the milk is to be gradually replaced by solid food. The excessive use of mineral waters is to be avoided. As regards the effect of drugs on the nephritis, opium and digitalis, given separately or together in small doses, are the only ones which have seemed to me to give any positive results. The regular use of the hot pack is of much service.

The anæmia is always marked, and an examination of the blood shows a diminution in the quantity of hæmoglobin and in the number of the red blood-cells. The most efficient treatment for this is the internal use of iron, the inhalation of oxygen, massage, out-door exercise, and the relief of constipation. In bad cases the anæmia cannot be improved.

The dropsy nearly always requires treatment. It is, as a rule, easier to get rid of it if the patients are kept in bed. Indeed, in a number of cases rest in bed and a milk diet will cause the dropsy to disappear without other treatment. Often, however, the dropsy is extensive and persistent; then we employ diuretics, cathartics, and diaphoretics, puncture the skin, and tap the serous cavities. It must be confessed that in some cases we are unable to control the dropsy at all.

The condition of the arteries and of the left ventricle of the heart must be watched throughout the disease. Contraction of the arteries calls for nitroglycerin, chloral hydrate, opium, or potassium iodide; a feeble heart demands digitalis, caffeine, or strophanthus.

The quantity of urea excreted daily tells us much concerning the progress of the nephritis.

When the patient passes into the condition of prostration, delirium, and stupor, with a feeble heart, we know that treatment is unavailing and that death will soon follow.

CHRONIC PRODUCTIVE (OR DIFFUSE) NEPHRITIS WITH EXUDATION.

Definition.—A chronic inflammation of the kidney attended with a growth of new connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, exudation from the blood-vessels, and sometimes changes in the walls of the arteries.

SYNONYMS.—Chronic Bright's disease; Chronic parenchymatous nephritis; Chronic glomerulo-nephritis; Waxy kidney; Large white kidney; Chronic diffuse nephritis; Interstitial nephritis; Chronic desquamative nephritis.

Although it is convenient to describe two forms of chronic nephritis—one with exudation and one without—yet it must be remembered that these are not separate lesions of the kidneys, but varieties of the same lesion. For in all these kidneys one form of inflammation—the productive, with the formation of new tissue—is present. The exudation from the vessels is something which is added to this, but does not change it.

In speaking of the exudation of serum from the vessels and its presence in the urine, we speak of it as it occurs during the whole course of the disease, and not as it occurs for short periods. We mean that in an exudative chronic nephritis there is usually a large quantity of albumin in the urine, but that in the protracted cases there may be periods during which the albumin diminishes or entirely disappears. In the same way, in a non-exudative nephritis there may be periods during which albumin is present in considerable quantities. Generally speaking, the character of the clinical symptoms will vary with the presence or absence of the albumin.

Etiology.—This form of nephritis occurs very frequently as a primary disease in young and middle-aged adults. It follows acute diffuse nephritis, chronic congestion, and chronic degeneration of the kidney. It may complicate syphilis, chronic phthisis, chronic endocarditis, prolonged suppuration, and chronic inflammation of the bones and joints.

Morbid Anatomy.—The kidneys are usually large, with smooth, roughened, or nodular surfaces. Less frequently they are small, with a white cortex, or may even resemble a normal kidney.

There is a very extensive growth of connective tissue in the cortex; the renal epithelium is swollen, granular, degenerated, fatty, broken, or flattened; the tubes contain coagulated matter, cast matter, or blood; the cortex tubes are atrophied in some places, dilated in others.

The glomeruli are changed in several different ways:

1. There is a growth of the capsule-cells in such numbers that they compress the tufts. The cells covering the capillaries are also increased in size and number. The capsule-cells may finally be changed into connective tissue and the tufts become atrophied.

2. The glomeruli are of large size; the cells covering the capillaries are increased in number, so that the outlines of the capillaries are lost, but yet the capillaries are not compressed nor the glomeruli atrophied.

3. There is a growth of the cells which cover the capillaries and of the cells within them. Of the cells which cover the capillaries, the cell-bodies become very large, the capillaries are compressed, and the glomeruli eventually become atrophied.

4. The walls of the capillary vessels become the seat of waxy degeneration, while the cells which cover them are increased in size and number.

5. If the nephritis follows chronic congestion, the capillaries are dilated, and there is an increase in the size and number of the cells which cover the capillaries.

The arteries remain unchanged, or they are the seat of obliterating endarteritis, or there is a symmetrical thickening of all the coats of the artery, or all the coats of the artery are thickened and converted into a uniform mass of dense connective tissue, or there is waxy degeneration of the walls of the artery.

Symptoms.—The urine varies in quantity at different times. When the nephritis is most quiescent the quantity of urine is normal. During the exacerbations of the nephritis the urine is scanty or suppressed. When the patients are doing badly, often when they are dropsical, the quantity of urine is very much increased.

The specific gravity and the proportion of urea to the ounce of urine slowly diminish. In the cases of shorter duration the specific gravity is apt to run between 1.012 and 1.020. In the very chronic cases it will be between 1.001

and 1.005. A very low specific gravity indicates a large growth of connective tissue in the stroma of the cortex or waxy degeneration of the capillaries of the glomeruli.

The urine always contains albumin and casts. During the active periods of the disease the quantity of albumin is very large; during its quiescent period it is smaller, and at times may entirely disappear. The number of casts varies in proportion to the quantity of albumin, but occasional exceptions to this rule are seen.

Dropsy may be considered a constant symptom of chronic exudative nephritis. It is rare to find a patient who goes through the disease without exhibiting it. It may be developed at any time in the disease, continue uninterruptedly, or occur only in attacks. A peculiar pallor of the skin and white color of the sclerotic is seldom absent, and is quite characteristic of the disease. It corresponds to a diminution in the quantity of hæmoglobin and in the number of the red blood-cells. These changes in the blood are not, as a rule, far advanced; but sometimes they are, and some cases even die with the symptoms of pernicious anæmia.

Many of the patients are troubled with headache and sleeplessness. Acute uræmic attacks, with contraction of the arteries, convulsions, etc., may occur in the course of a chronic exudative nephritis, but they are of very much more frequent occurrence with the non-exudative form of the disease.

Chronic uræmia, on the contrary, is one of the ordinary ways in which an exudative nephritis proves fatal. The patients pass into a condition of alternating delirium and stupor, with a rapid, feeble, soft pulse.

Simple neuro-retinitis or nephritic retinitis are developed in a moderate number of cases.

Dyspnœa is a nearly constant symptom, but it is not always the same kind of dyspnœa nor always produced by the same cause. It may be due to hydrothorax, to œdema of the lungs, to contraction of the arteries, or to failure of of the heart's action.

In many patients the dyspnœa due to contraction of the arteries or to failure of the heart's action is the first symptom which attracts attention. It is a dyspnœa which comes on in attacks, especially at night and in the early morning, and is worse when the patient lies down. It often begins while the patient is apparently in good health, but it is a sure premonition of serious disease.

A catarrhal bronchitis with cough and expectoration is often present.

Loss of appetite, nausea, and vomiting are frequent symptoms.

The heart is often affected. There may be hypertrophy of the left ventricle, dilatation of both ventricles, chronic endocarditis, myocarditis, or a feeble heart.

Course of the Disease.—There is hardly any limit to the variations of the disease, but the most constant symptoms are anæmia, dropsy, and albumin in the urine.

1. There are cases in which the symptoms are nearly continuous; the patients

get steadily worse, and die within one or two years. The anæmia, the dropsy, and the albumin are constantly present, and the patients die with dropsy or with chronic uræmia.

2. There are cases in which the anæmia, the dropsy, and the dyspnœa come on in attacks which last for weeks or months. Between the attacks the patients are comparatively well, often able to work, although the urine always contains albumin.

3. There are cases in which a number of years before death the patient has an attack of dropsy, etc., from which he apparently recovers and goes on able to work, but with urine of low specific gravity, which sometimes contains albumin. After an interval of many years comes the fatal attack, with all the characteristic symptoms.

4. There are cases which for years have no symptoms but pallor of the skin and urine of low specific gravity containing albumin. These patients often for a long time feel so well that they cannot understand that they have a serious disease.

5. There are cases in which the first symptom is the attack of spasmodic dyspnœa, the patients otherwise feeling well. It may be months or years before the other symptoms are developed.

6. There may be a history of chronic endocarditis lasting for years before the renal symptoms are developed.

7. Some cases apparently recover from the disease.

The **prognosis** is bad, but life may be prolonged for many years with but few symptoms, and recovery seems to be possible.

Treatment.—As the urine of these patients contains less than the normal quantity of urea, they need to pass more than the normal quantity of urine; about sixty ounces of urine a day seems to be the proper quantity for them. Any considerable diminution or increase in the quantity of urine is to be rectified by regulating the diet and the ingestion of fluids, the action of the heart, and the contraction of the arteries.

For the relief of the dropsy we use rest in bed, limitation of the quantity of fluids taken, cardiac stimulants or arterial dilators, diuretics, cathartics, or diaphoretics. It may be necessary to puncture the skin or to tap the serous cavities.

The anæmia requires from time to time the use of iron.

If the patients are troubled with headache and sleeplessness, the treatment depends upon the condition of the heart and arteries. With a feeble heart and dilated arteries we use the cardiac stimulants. With contracted arteries we use nitro-glycerin, chloral hydrate, potassium iodide, or opium.

Attacks of general convulsions, muscular twitchings, or coma usually accompany contraction of the arteries, and are to be treated by the arterial dilators.

The condition of alternating delirium and stupor, with or without muscular twitchings and convulsions, but with dilated arteries, is not easily benefited by treatment. The most efficient measure is the daily use of the hot pack.

The dyspnœa which accompanies contracted arteries requires the use of the arterial dilators; that which is due to a feeble heart calls for the cardiac stimulants; that which depends upon œdema of the lungs or fluid in the pleural cavities is met by the treatment for dropsy. Contraction of the arteries, which continues in spite of the use of the arterial dilators, can sometimes be relieved by calomel.

The loss of appetite, nausea, and vomiting may call for a fluid diet, the use of the alkalies or of oxalate of cerium, sometimes for the washing out of the stomach.

As regards the general management of the patients, the rule is that, when the dropsy and other symptoms are pronounced, the patients are to be kept in bed and on a fluid diet, but that whenever it is possible they should have solid food and an out-of-door life. The good effects of a prolonged residence in a warm, dry climate are often very marked.

CHRONIC PRODUCTIVE (OR DIFFUSE) NEPHRITIS WITHOUT EXUDATION.

Definition.—A chronic inflammation of the kidney attended with a growth of new connective tissue in the stroma, permanent changes in the glomeruli, degeneration of the renal epithelium, and sometimes changes in the walls of the arteries.

SYNONYMS.—Chronic Bright's disease; Cirrhosis of the kidney; Granular degeneration; Interstitial nephritis; Chronic indurative nephritis; the Arterio-sclerotic kidney.

Etiology.—This form of nephritis belongs to persons over twenty years of age. It seems to be caused by chronic alcoholism, chronic lead-poisoning, gout, and by the same conditions as those which cause emphysema, endocarditis, endarteritis, and cirrhosis of the liver. It follows chronic congestion of the kidney, hydronephrosis, and chronic pyelitis.

Morbid Anatomy.—The larger number of the affected organs are found after death to be diminished in size; the two kidneys together may not weigh more than two ounces. The capsules are adherent, the surfaces are roughened or nodular, the cortex is thin and of a red or gray color. A considerable number do not differ in their size or appearance from normal kidneys, except that the capsules are adherent and the surfaces roughened. A few of these kidneys are large, weighing together from sixteen to thirty-two ounces, with smooth or nodular surfaces and a cortex of red, gray, or white color. If the nephritis follows chronic congestion, the kidneys remain hard, but the cortex becomes thinned, the capsules adherent, and the surface roughened.

There is a growth of new connective tissue in the cortex, and also in the pyramids, which becomes more and more marked as the disease goes on. In the cortex the new tissue follows the distribution of the normal subcapsular areas of connective tissue, is in the form of irregular masses, or is distributed diffusely between the tubes. In the pyramids the growth of new connective tissue is diffuse.

The tubes, both in the cortex and pyramids, undergo marked changes. Those included in the masses of connective tissue are diminished in size; their epithelium is flattened; some contain cast matter, many are obliterated. The tubes between the masses of new connective tissue are more or less dilated; their epithelium is flattened, cuboidal, swollen, degenerated, or fatty. The dilatation of the tubes may reach such a point as to form cysts of some size, which contain fluid or coagulated matter. These cysts follow the lines of groups of tubes or are situated near the capsules.

Of the glomeruli a certain number remain of normal size, but with the tuft-cells swollen or multiplied. Many others are found in all stages of atrophy, until they are converted into little fibrous balls. The atrophy seems to depend partly on the growth of tuft-cells and intracapillary cells, partly on the thickening of the capsules, partly on the occlusion of the arteries. If the chronic nephritis follows chronic congestion, the glomeruli remain large, but with a marked growth of tuft-cells; or they become atrophied, but with the dilatation of the capillaries still evident. The capillaries of the glomeruli may be the seat of a waxy degeneration.

The arteries exhibit the same changes as are found in chronic exudative nephritis.

Complicating Lesions.—Hypertrophy of the left ventricle of the heart is frequently caused by exudative nephritis, but still more frequently by the non-exudative form; but it must be admitted that such an hypertrophy, although frequent, is not constant—that both with exudative and non-exudative nephritis there may be no change in the wall of the left ventricle. The hypertrophy of the wall of the ventricle may after a time be succeeded by dilatation, or chronic degeneration, or myocarditis.

Chronic endocarditis is very frequently associated with chronic Bright's disease. The valvular lesions may cause chronic congestion, chronic degeneration, or chronic nephritis; or the same patient may suffer from chronic endocarditis and either form of chronic nephritis, the lesions associated possibly due to the same causes, but not dependent on each other.

Pulmonary emphysema, chronic endarteritis, and cirrhosis of the liver, all of them examples of chronic productive inflammation, frequently accompany chronic nephritis.

With the endarteritis comes the additional danger of cerebral hæmorrhage.

Patients suffering with chronic nephritis are more liable than are other persons to attacks of pericarditis, bronchitis, and gastric catarrh.

Symptoms.—The typical urine of chronic non-exudative nephritis is a urine increased in quantity, of a specific gravity of about 1.010, containing a diminished quantity of urea, without albumin or casts, or with a trace of albumin and a very few casts, except during exacerbations of the nephritis, when the quantity of albumin and the number of casts may be considerable. Very important modifications of the urine are, however, of ordinary occurrence. It is quite possible, with nephritis of this type far advanced, to have urine not below 1.023 in specific gravity, and without albumin or casts. In such cases the diagnosis has to be made without reference to the urine. On the other hand, there are cases in which the specific gravity of the urine falls very low, almost to 1.000, either with or without waxy degeneration of the vessels. There are cases in which the quantity of urine is very much increased, several quarts in the twenty-four hours. During the attacks of contraction of the arteries, to which these patients are liable, the urine may be diminished to a few ounces or even suppressed.

In a great many of the cases cerebral symptoms are developed at some time in the course of the disease. Headache and sleeplessness are often present, the headache sometimes so severe and continuous that the patient is nearly maniacal; or instead of the headache there are neuralgic pains in different parts of the body.

Muscular twitchings and general convulsions are much more serious; they may be an early symptom or not occur until late in the disease.

Hemiplegia, with or without aphasia, may be the first symptom to call attention to the nephritis, or may not occur until late in the disease. The invasion of the hemiplegia is sudden, and is usually accompanied by coma. There is loss of motion alone or of both motion and sensation. The hemiplegia, coma, and aphasia may continue up to the time of the patient's death or disappear after a few hours or days. In the latter case the patient may have several such attacks. These attacks have been ascribed to localized œdema of the brain. In the cases which I have seen there were no changes in the brain-tissue, but the cerebral arteries were damaged by chronic endarteritis.

Delirium, mild or violent, stupor, and coma may come on in sudden attacks or be developed slowly and gradually.

When these cerebral symptoms come on in attacks, the arteries are contracted, the temperature is raised, and the patients are said to suffer from acute uræmia. Very often they recover from a number of these attacks. In the fatal attacks the pulse often loses its tension, but becomes rapid and feeble, and the patients die comatose, with a feeble heart.

Instead of such acute attacks of cerebral symptoms, delirium and stupor may come on gradually in persons already much reduced by the kidney disease. The temperature is then apt to be below the normal, and the pulse is rapid and feeble.

Temporary blindness, neuro-retinitis, or nephritic retinitis are developed in a moderate number of these patients.

Chronic bronchitis and emphysema very frequently exist, and their symptoms often form a large part of the clinical history.

The left ventricle of the heart is constantly hypertrophied. This by itself gives no symptoms; but if the arteries become contracted, or if the hypertrophied heart becomes feeble, then disturbances of the circulation are established which cause serious symptoms.

In the same way, the complicating endocarditis which so often exists gives no trouble until the valves are a good deal changed, or the ventricles dilated, or the heart's action altered, or the arteries contracted ; then the circulation is interfered with, and the results of venous congestion of different parts of the body show themselves.

Dyspnœa is a very frequent symptom, often the first symptom noticed by by the patient. It is a spasmodic dyspnœa coming on in attacks which last for minutes, hours, or days. It is made worse by mental or bodily exertion or by the recumbent position. It does not resemble pulmonary asthma. It is apparently due to the association of changes in the arteries and in the heart. With contraction of the arteries alone or with a feeble heart alone no dyspnœa may exist; but if the contraction of the arterties be so great that the hypertrophied and laboring heart is unable to overcome the obstruction, or if with the contracted arteries the heart become dilated or feeble, then the attacks of dyspnœa begin. At first the attacks are not severe and are of short duration, but if the mechanical conditions which cause them cannot be controlled, they become longer and more distressing.

Perhaps the most striking examples of this dyspnœa are in the patients in whom it is the first symptom of the nephritis. They are apt to be middle-aged or elderly men, often engaged in large financial or commercial enterprises. They profess that they feel quite well and that they can attend to their affairs perfectly, but that they are very much annoyed because early every morning they have an attack of asthma. In spite of their professions of good health, it is evident that they are pale and that they have dyspnœa on exertion. The heart is found to be enlarged, with or without a murmur ; its action is either labored or feeble. The pulse is tense. The urine is of a specific gravity of 1.010 to 1.030 ; it contains no albumin or only a trace. In the earlier stages of the disease this dyspnœa can be controlled, but later on it is more distressing and difficult to remedy.

The stomach may become the seat of catarrhal gastritis or of spasmodic vomiting; but in some patients it continues to perform its functions fairly well.

Dropsy, as a rule, is absent with non-exudative nephritis, unless it be complicated by chronic endocarditis or cirrhosis of the liver.

Profuse bleeding from atrophied kidneys has been described by Bowlby in three cases.

In all cases, after a time, the nephritis exerts its effects upon the nutrition of the patient, and the flesh and strength are diminished. On the other hand, the patients do not become as pale as they do in exudative nephritis.

Course of the Disease.—It is characteristic of the chronic productive inflammation of the lungs, the heart, the arteries, the liver, and the kidneys that, while they often exist as serious and fatal diseases, they also frequently exist as lesions that do not interfere with general good health and long life. This seems to depend, in a part at least, on the rapidity with which the inflammatory changes in these different parts of the body are developed. If they are developed slowly enough, the functions of the organ continue to be performed, in spite of the new growth of connective tissue in it.

So, with the kidneys, it is common enough to find chronic non-exudative nephritis far advanced in persons who die from accident or intercurrent disease, and have never given symptoms of renal disease.

In the same way, we can often observe for years persons who have urine of low specific gravity, an hypertrophied left ventricle of the heart, and occasionally some increase of tension in the arteries, and yet who habitually enjoy very fair health.

Yet these same persons, if they are attacked with pneumonia or pericarditis or suffer from a severe accident, will often develop serious or even fatal renal symptoms.

A very common form for the disease to take is that of attacks which are repeated a number of times, each attack worse than the preceding, and the general health more and more impaired between the attacks. During the attacks there are cerebral symptoms more or less severe—headache, sleeplessness, delirium, stupor, coma, convulsions. Dyspnœa may be present or absent. The arteries are contracted, the pulse tense. There is loss of appetite, nausea, and vomiting. The urine is of low specific gravity and usually contains albumin. Between the attacks the patients at first seem to be fairly well, but later they gradually lose flesh and strength. The urine between the attacks is of low specific gravity and contains little or no albumin. They finally die in one of the attacks, feeble and emaciated.

In some of the patients spasmodic dyspnœa is the first symptom. This can often be controlled for months or years, and the patients then seem to be well. After a time, however, it is more difficult to manage, and other renal symptoms are added.

In some cases there are no symptoms for a long time, so that persons apparently in good health are attacked without warning by convulsions, coma, delirium, or hemiplegia. They may die in the first attacks or live to go through subsequent ones.

In some cases the only symptoms up to the time of the patient's death are gradual loss of flesh and strength and disturbances of digestion, the patient dying feeble and emaciated. These cases are hard to diagnosticate, unless the specific gravity of the urine be low and a little albumin present. Otherwise there is nothing to draw attention to the kidneys as the cause of the illness. Loss of eyesight from nephritic neuro-retinitis may be the first symptom.

The patients may suffer from the symptoms of cardiac disease for years before congestion or degeneration of the kidney is succeeded by chronic nephritis.

In many cases the course of the nephritis is modified by the complicating emphysema, phthisis, endocarditis, endarteritis, or cirrhosis of the liver.

In speaking of the different forms of nephritis it has been necessary to speak of the symptoms dependent on contraction of the arteries and of the condition of chronic uræmia. These two conditions are entirely distinct, yet they are often described under the names of acute and chronic uræmia. I think that it is very important to understand these two conditions as definitely as possible.

Contraction of the arteries (acute uræmia) causes involuntary contractions of groups of muscles, general convulsions, stupor, coma, dyspnœa, labored heart-action, hypertrophy of the left ventricle of the heart, blindness, aphasia, hemiplegia, a high temperature, and perhaps dropsy. Its existence can be readily appreciated by the character of the pulse and of the action of the heart.

It occurs not only with the different forms of nephritis, but also with angina pectoris, pulmonary emphysema, and chronic endarteritis.

In nephritis it is often developed while the specific gravity of the urine is still good, and the quantity of the urine is not diminished until after the contraction of the arteries is established. There can be no reason, therefore, to believe that the contraction of the arteries is due to contamination of the blood by excrementitious substances, and we must frankly admit that the reason of the contraction is as yet unknown. We know, however, by a wide experience that, when by the use of drugs we can dilate the arteries, the symptoms dependent upon their contraction will disappear.

In chronic uræmia the convulsions, sudden coma, dyspnœa, high temperature, aphasia, hemiplegia, and contraction of the arteries are absent. The action of the heart and the pulse are feeble. The patients pass into a condition of great prostration with alternating delirium and stupor.

This condition is developed in persons who are passing little or no urine or urine of low specific gravity. It evidently is caused by the contamination of the blood by excrementitious substances.

Treatment.—The progress of the nephritis can apparently be favorably affected by attention to the diet and mode of life and to climate. As regards the diet, the quantity of sugars and starches taken should be restricted and the ingestion of fats encouraged. The use of wine, spirits, and tobacco should be discontinued. Exercise in the open air is to be advised as long as the strength permits of it. As regards climate, we must consult the idiosyncrasy of the patient; it should be a climate where he eats well, sleeps well, and feels well. There is a decided advantage in not remaining in the same place throughout the year.

Disturbances of the circulation, with dyspnœa, vomiting, or cerebral symptoms, are liable to come on at any time. In these cases, therefore, we constantly watch the heart and arteries. If the arteries become contracted and the pulse tense, we at once give potassium iodide, nitro-glycerin, chloral hydrate, or opium. If the heart's action becomes feeble, we use digitalis, caffeine, or strophanthus. As regards the use of opium, it is apparently safe and beneficial to use it if the arteries are contracted; if they are not, even a very little opium may cause poisoning and death. So with general convulsions or sudden coma, hypodermic injections of from one-eighth to one-fourth of a grain of morphine may be of decided benefit; and in protracted contraction of the arteries, with sleeplessness and restlessness, one-eighth of a grain of morphine every three hours may give great relief.

PUERPERAL ECLAMPSIA.

It is well known that women in the later months of pregnancy, during labor, and immediately after childbirth may develop cerebral symptoms, dropsy, and anæmia. These morbid conditions are especially frequent in primiparæ, in young women, and with twin pregnancies. They may be repeated in several successive pregnancies. They belong to the second half of pregnancy, increase in severity as the pregnancy advances, and are at their worst during labor.

Morbid Anatomy.—In the fatal cases it has been found that some of the kidneys show dilatation of their pelves and ureters, some are in the condition of acute degeneration, some are examples of acute exudative nephritis, some of acute productive nephritis, while in some the kidneys appear to be normal. Even in young women the cerebral arteries may be diseased. In one case I found clots of blood in the third and fourth ventricles of the brain.

Symptoms.—The symptoms occur before, during, and after labor. Rosenstein gives 121 cases before, 260 during, and 118 after labor.

In the cases without disease of the kidney the patients are anæmic, the veins throughout the body contain too much blood, the arteries are contracted, there are muscular twitchings, general convulsions or coma, the urine is scanty; after the convulsions the latter may contain albumin.

In the cases with exudative or productive nephritis the only symptoms may be scanty and albuminous urine; or scanty and albuminous urine with dropsy; or scanty and albuminous urine with anæmia, nausea and vomiting, headache, blindness, muscular twitchings, convulsions, coma, or hemiplegia. Some of the patients recover; others die in convulsions, coma, or hemiplegia; others live but suffer from chronic nephritis.

Treatment.—While there have been many different opinions as to the nature of puerperal eclampsia, there is a good deal of uniformity as to the treatment. If the convulsions come on at about the natural end of pregnancy, it is generally agreed that labor should be brought on and the child delivered as soon as possible. Apart from this, the ordinary treatment is to unload the veins and dilate the arteries. This is effected by venesection and by the use of nitro-glycerin, chloral hydrate, opium, and chloroform.

In order to guard against the puerperal eclampsia it is of more practical use to watch the arteries and heart than to test the urine for albumin.

SUPPURATIVE NEPHRITIS.

Suppurative inflammation of the pelvis of the kidney and of the kidney itself occurs under several different conditions: It is the result of injuries; it is due to emboli; it occurs without discoverable cause; it is secondary to cystitis, the cystitis being due to strictures of the urethra, to stone in the bladder, to paraplegia, to operations on the urethra, bladder, and uterus, to gonorrhœa, or to enlarged prostate. Chronic suppurative pyelo-nephritis is often caused by the presence of calculi in the pelvis of the kidney.

1. Suppurative Nephritis from Injury.—Gunshot wounds, incised or punctured wounds, falls, blows, and kicks, are the ordinary traumatic causes. If the injury be a very severe one, it causes the death of the patient in a short time; if it be less severe, suppurative inflammation may be developed.

The inflammatory process may be diffuse, so that the whole of one or both kidneys is converted into a soft mass composed of pus, blood, and broken-down tissue; or it is circumscribed, and one or more abscesses are found in the kidney which may communicate with its pelvis.

Symptoms.—Rigors mark the beginning of the suppuration, and are often repeated through its course. A febrile movement is developed which is apt to assume the hectic character with sweating. There is often vomiting. There may be very severe pain referred to the region of the inflamed kidneys. The urine is diminished or suppressed; it contains blood alone or blood and pus. In the bad cases the patients pass into the typhoid condition, become delirious, and die comatose or with a very rapid and feeble pulse. Or the disease is protracted, the patients become more and more emaciated, and finally die exhausted. In other cases the symptoms abate, the urine returns to its natural condition, and the patients recover.

Treatment.—The management of these cases is rather surgical than medical. The external wound is to be treated antiseptically, and the suppurative kidney is to be incised or removed, as may be necessary.

2. Abscesses produced by Emboli.—In ordinary endocarditis with vegetations on the valves it often happens that fragments of the vegetations become fixed in the branches of the renal arteries. When this is the case white infarctions are produced.

With malignant endocarditis and with septicæmic infections emboli find their way into the branches of the renal artery and set up circumscribed foci of suppurative inflammation. The kidneys become enlarged, and are studded with little white points surrounded by red zones. These little white points are formed by an infiltration of pus-cells between the tubes, followed by the death and breaking down of the kidney-tissue. The bacteria of suppuration are found in these little abscesses.

Symptoms.—These embolic abscesses can hardly be said to have a clinical history. Whatever symptoms may belong to them are lost in those of the general disease from which the patient is suffering.

3. Idiopathic Abscesses.—These occur without discoverable cause. Only one kidney is involved. We find after death part of the kidney destroyed; the remaining portions contain abscesses; the pelves are dilated and contain pus, the capsules are thickened, the suppurative inflammation may extend to the surrounding tissues so that sinuses are formed, and even perforations into the intestine or through the diaphragm. It is very difficult in these cases to tell whether the inflammation begins in the kidney or in its pelvis.

Symptoms.—The symptoms begin gradually and are for some time obscure.

There are repeated chills and irregular febrile movement. The patients lose flesh and strength, become anæmic, and are often troubled by nausea and vomiting. There is more or less pain over the inflamed kidney. After a time the pelvis of the kidney may be so much dilated as to form a tumor. If the pus escapes from time to time through the ureter, this tumor will vary in size. The urine at intervals contains pus and fragments of broken-down kidney-tissue. If the suppurative inflammation extends, there will be sinuses running behind the peritoneum, or into the colon, or upward through the diaphragm. The disease is apt to last a long time. The patients are liable to have a chronic nephritis of the other kidney or waxy degeneration of the viscera.

Treatment.—The only plan of treatment is to cut down on the suppurating kidney and treat it as an abscess, or to remove it altogether.

4. Suppurative Pyelo-nephritis with Cystitis.—Both kidneys become inflamed. The pelves are congested and coated with pus or fibrin. The kidneys are swollen, congested, and studded with foci of pus. The smallest foci are not visible to the naked eye, but with the microscope we find collections of pus-cells between the tubes, with swelling and degeneration of the epithelium within the tubes. The larger purulent foci look like white streaks or wedges running parallel to the tubes and surrounded by zones of congestion. The larger abscesses replace considerable portions of the kidney.

The ureters in some cases are inflamed, their walls thickened, their inner surfaces coated with pus or fibrin. The bladder presents the lesions of acute or chronic cystitis.

Etiology.—This form of nephritis seems to be always secondary to a cystitis, the infection extending from the bladder through the ureters to the kidneys. The cases of cystitis in which a suppurative nephritis is likely to be developed are those due to strictures of the urethra, stone in the bladder, operations on the urethra, bladder, and uterus, paraplegia, gonorrhœa, and enlarged prostate.

Symptoms.—When the nephritis occurs with cystitis due to stone in the bladder, strictures, or operations on the genito-urinary tract, the symptoms are much the same. The patient has first the symptoms belonging to the cystitis; then he is attacked with chills followed by a febrile movement. The chills are repeated; the febrile movement is irregular and accompanied by profuse sweating. There is a rapid change in the general condition of the patient; he becomes more and more prostrated and emaciated from day to day. The face is drawn and anxious, the tongue dry and brown, the pulse rapid and feeble; delirium is developed, and the patient finally dies in the septic condition. The urine is diminished in quantity or suppressed; it contains blood, pus, and mucus derived partly from the bladder, partly from the kidneys.

Cases of suppurative nephritis complicating gonorrhœa are fortunately not common, but several of them have been observed. Murchison describes two cases, in both of which the cerebral symptoms were very marked—delirium, convulsions, and coma. I have seen one such case. The patient was a prostitute who came into the hospital with a specific vaginitis. After a few days she developed the symptoms of an acute cystitis; then after a few more days she was attacked with chills and a febrile movement, passed rapidly into the septic condition, and died. At the autopsy there were found acute cystitis, pyelitis, and numerous small abscesses in both kidneys.

When suppurative nephritis complicates the cystitis due to enlarged prostate, the symptoms are somewhat different. The patients are usually men over fifty. They have generally suffered from the symptoms of enlarged prostate, retention of urine, either constant or intermittent, and more or less cystitis, with pus and mucus in the urine. Sometimes, however, no such history is obtained; the patients assert that they have had no previous bladder trouble. The first symptom is a diminution in the quantity of urine, with the appearance of blood mixed with it, or the urine may be suppressed altogether. The blood is present in considerable quantities, so that the patients may seem to pass pure blood instead of urine. The patients rapidly become prostrated and very anxious. There are usually no chills, and there may be no febrile movement. The prostration becomes more marked, the pulse is rapid and feeble, the skin cold and bathed in perspiration, and the patients die in collapse at the end of a few days. Or, instead of such a history, the patients may behave as if they were the subjects of septic poisoning.

Prognosis.—Suppurative nephritis secondary to cystitis is a very fatal disease: so far as I know, all the cases die.

Treatment.—The treatment for these cases is altogether a preventive one, directed to the cystitis. When the nephritis is once established we have no further control over the case.

Tubercular Nephritis.

The different portions of the genito-urinary tract—the kidneys, ureters, bladder, seminal vesicles, prostate, testicle, uterus, and ovaries—may become the seat of a localized tubercular inflammation.

Such an inflammation may involve one of these organs or several. If several are involved, they are on the same side of the body, usually the left side. The inflammation is attended with the presence of tubercle bacilli and the formation of tubercles. The new tubercular tissue soon dies and undergoes cheesy degeneration.

In the kidneys the inflammation begins in the mucous membrane of the pelves and calyces, and extends to the parenchyma until a large part of the organ is replaced by the degenerated new tissue. The cheesy masses may soften or become calcified, while the kidney between them is converted into fibrous tissue more or less infiltrated with pus.

The other kidney, after a time, is apt to become the seat of the exudative form of chronic nephritis, with waxy degeneration of the blood-vessels.

The tubercular nephritis may be complicated by tubercular inflammation of other parts of the genito-urinary tract on the same side of the body, by tubercular peritonitis, pulmonary tuberculosis, or general tuberculosis.

The disease is said to occur at all ages; it is most frequent in middle-aged persons. It occurs twice as often in men as in women.

Symptoms.—The urine usually, but not always, contains from time to time blood, pus, detritus, epithelium, shreds of tissue, and tubercle bacilli. When the other kidney has become the seat of chronic nephritis, the specific gravity of the urine falls and albumin and casts are present.

Pain, either continuous or in paroxysms, and tenderness are often present over the inflamed kidney. There may be hectic fever with night-sweats; the patients gradually lose flesh and strength. The kidney may become enlarged so as to form a tumor which can be felt. After a time there are added the symptoms of tubercular inflammation of other parts of the genito-urinary tract, of tubercular peritonitis, of pulmonary phthisis, of waxy degeneration of the viscera, or of the chronic nephritis of the remaining kidney.

The disease lasts, as a rule, for several years. Most of the cases terminate fatally, but it is possible for the inflammation to stop and for the patient to recover.

Treatment.—The proper treatment for tubercular nephritis ought to be the removal of the diseased kidney. The practical difficulty is to make the diagnosis before other parts of the genito-urinary tract have become tubercular or before the remaining kidney has become the seat of chronic nephritis.

We may hope that climate and feeding may have the same good effects on tubercular nephritis as on pulmonary tuberculosis.

PERINEPHRITIS.

The loose connective tissue around the kidney may become the seat of suppurative inflammation, and in this way abscesses of considerable size may be formed.

Etiology.—Perinephritis is either secondary or primary. The secondary cases are due to extension of the inflammation from abscesses in the vicinity, such as are formed with caries of the spine, pelvic cellulitis, puerperal parametritis, perityphlitis, suppuration of the kidney, and pyelo-nephritis. The primary cases occur after exposure to cold, after contusions over the lumbar region, great muscular exertion, and without discoverable cause. The lesion is said to complicate typhus and typhoid fever and small-pox. The disease occurs both in children and adults; most of the cases reported have been between the ages of twenty and forty years.

Morbid Anatomy.—The connective tissue behind the kidney seems to be the usual point of origin of the inflammatory process, and it is here that the pus first collects. After the abscess has reached a certain size the suppuration seems to have a natural tendency to spread, and the pus burrows in different directions—backward through the muscles; downward along the iliac fossa, even as far as the perineum, scrotum, or vagina; forward into the peritoneal cavity, the colon, or the bladder; upward through the diaphragm. The kidney is compressed by the abscess or becomes involved in the suppurative process. The soft parts around the abscess become thickened and indurated.

Symptoms.—The disease begins, as a rule, with pain and tenderness referred to one lumbar region, between the lower border of the ribs and the crest of the ilium, sometimes to a point above or below this. At about the same time are developed repeated rigors, a febrile movement with evening exacerbations, sweating, loss of appetite, vomiting, and prostration. These are all the symptoms for from one to two weeks. Then the skin over the lumbar region on one side becomes red and œdematous; the corresponding thigh is kept flexed and rigid, for any movement of it gives pain. Then the lumbar region becomes more and more swollen until fluctuation can be made out, and finally the abscess breaks through the skin. If such cases are left to run their course, the abscess may reach a very large size. If the pus does not extend backward, but in some other direction, the symptoms are more obscure, for the local symptoms of an abscess in the back are absent.

If the abscess ruptures into the peritoneal cavity, the symptoms of acute general peritonitis are suddenly developed. If it perforates into the colon or bladder, the pus is discharged with the fæces or the urine. If the perforation is through the diaphragm, there will be empyema, or the lung becomes adherent and pus is coughed up from the bronchi. As soon as the abscess is opened and the pus escapes, the acute constitutional symptoms subside.

Trousseau believes that the inflammatory process sometimes stops short of the production of pus. In such cases, of course, there are no evidences of the formation of an abscess.

If the abscess ruptures spontaneously or is opened by the surgeon, the patient is likely to recover, but the suppurative process may continue and the patient die exhausted, usually with waxy viscera.

Perforation into the peritoneum, the pleura, or the lung causes death.

Treatment.—The main point in the treatment is to discover the abscess and to open it. The longer the suppuration goes on and the larger the abscess, so much the worse is the prognosis. It is proper to explore with the aspirator after the disease has lasted for a few days, even if no fluctuation can be made out.

HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis and calyces of the kidney.

Etiology.—Dilatation of the pelvis and calyces of one or of both kidneys can be produced by any mechanical obstruction to the escape of urine.

Such an obstruction may begin during fœtal life, so that when the child is born both ureters and the pelves of both kidneys are found much dilated. Such children die soon after birth. The hydronephrosis in these cases is due to some congenital malformation, but occasionally we see this condition in children in whom it is very difficult to find the seat of obstruction. It is supposed that in these cases there exists a membranous obstruction which is broken by the probe used to explore the urethra.

In adults the mechanical cause of the obstruction to the escape of urine may be situated in the urethra, bladder, or uterus, or in the abdominal cavity near the ureters. According to the position of the obstruction, either one or both kidneys are affected.

Morbid Anatomy.—The pelvis and calyces of the affected kidney are more or less dilated ; the mucous membrane is thin and shining or thickened. The kidney-tissue becomes more and more thinned as the dilatation goes on, and after a time in the thinned kidney a chronic nephritis is set up. Then there is a growth of new connective tissue in the stroma of the pyramids and cortex ; in some of the glomeruli there is a growth of the cells covering the capillaries, while other glomeruli are atrophied ; degeneration of the renal epithelium and thickening of the walls of the arteries are also present.

Symptoms.—The patients suffer for a considerable length of time from the inconveniences belonging to the retention of urine. If the retention be due to stricture of the urethra or to disease of the bladder, the entire history is straightforward. Obstructions of the ureters, however, may give no evidence of their existence, and we find after death strictures of one or both ureters for which we are unable to account.

It is not uncommon to find after death well-marked hydronephrosis of one or both kidneys in patients who have had no renal symptoms at all.

In some cases, after a time, there is developed in the compressed kidneys a chronic productive nephritis with a little exudation. Then the specific gravity of the urine gradually falls ; a small quantity of albumin and a few casts make their appearance. The quantity of the urine remains normal, or is increased, or diminished, or suppressed.

The patients may at any time have contraction of the arteries with an elevated temperature and cerebral symptoms, or they may pass into the condition of chronic uræmia with a feeble pulse and low temperature.

In other cases the most prominent symptom is the presence of a tumor in the abdominal cavity. The disease is then usually confined to one kidney. The tumor lies in the region of the loins, extending upward, downward, and forward as it increases in size, and pushing the intestines forward and to one side. The surface of these tumors may become inflamed, and adhesions formed to the surrounding tissues As they become larger they give rise to much discomfort and pain. The tumors feel like cysts ; from time to time they may diminish in size, with an increased flow of urine from the bladder ; they are not movable If the disease is caused by renal calculi, there may be well-marked attacks of renal colic with bloody urine. If the fluid from the tumor be drawn off, it usually contains urinary salts, but sometimes it is only clear serum.

In still other cases of hydronephorosis a suppurative inflammation attacks the pelvis of the kidney, and the patient suffers from pyelitis.

Treatment.—The treatment of hydronephrosis is altogether surgical. If possible, the cause of the retention of urine must be relieved. If large cysts are formed by the diseased kidney, they must be opened or the kidney removed.

PYELITIS.

It has already been mentioned, in describing suppurative nephritis, that with chronic or acute cystitis there may be an extension of the inflammation from the bladder to the pelves of the kidneys, with suppurative inflammation of the pelves and of the kidneys themselves. In this form of acute pyelitis the inflammation of the kidney is more important than that of the pelves.

Calculi in the pelvis of the kidney often set up a pyelitis, which will be described with the account of the renal calculi.

An acute catarrhal pyelitis is said to be caused by the use of turpentine and cantharides, and by typhoid fever and the exanthemata. It lasts but a short time, and gives rise to no symptoms of importance.

The important form of pyelitis is the chronic inflammation which most frequently follows cystitis, sometimes succeeds hydronephrosis, and rarely seems to be a primary inflammation. While in most cases the pyelitis is clearly secondary to the cystitis, it may very well happen that the bladder will get well, while the pyelitis continues. In the cases of pyelitis which follows pregnancy it is not always easy to say whether the inflammation is due to the temporary hydronephrosis or to the cystitis produced by dirty catheters.

Morbid Anatomy.—The mucous membrane of the pelvis and calyces is thickened, its stroma more or less infiltrated with cells, the layer of epithelium thickened in some places, thinned in others, its surface coated with mucus or muco-pus. The pelvis and calyces are more or less dilated; they may contain uric-acid or oxalate-of-lime calculi, which have caused the inflammation; or phosphatic concretions, which are the result of it. In the kidney itself there may be a growth of connective tissue in the stroma of the pyramids and cortex, with degeneration of the epithelium and atrophy of the glomeruli, or a suppurative inflammation which may destroy considerable portions of the kidney.

Symptoms.—In the majority of the patients the symptoms of the cystitis, of the enlarged prostate, or of the stricture of the urethra are prominent features of the case.

So far as the pyelitis is concerned, the patients have pain and tenderness referred to the position of the diseased kidney. The urine from time to time contains blood, mucus, and epithelial cells from the pelvis of the kidney; later in the disease many pus-cells are mixed with the urine, and sometimes phosphatic concretions. The urine is more frequently acid than is the case with cystitis. The pus may be discharged continuously, or the ureter may from time to time become occluded, and the urine coming from the other kidney will be clear until the obstruction is overcome; then a large quantity of purulent urine will again be discharged.

In some patients the purulent inflammation of the pelvis continues; more or less suppurative inflammation of the kidney is added; there are also progressive loss of flesh and strength, hectic fever, chills, waxy degeneration of the viscera; and the patients finally die exhausted by the disease.

The distention of the pelvis may be so great as to form a fluctuating, painful tumor of considerable size. If the ureter be not entirely occluded, the pus will escape from time to time, with a corresponding change in the size of the tumor.

Occasionally, after a time the purulent discharge ceases, the pelvis contracts, the kidney is atrophied, and the patients get well with only one useful kidney. In some of the cases the secondary nephritis after a time gives symptoms. The specific gravity of the urine falls. The patients lose flesh and strength, become anæmic, have more or less dropsy, and finally have acute cerebral symptoms or pass into the condition of chronic uræmia.

If phosphatic concretions are formed in the pelvis, fragments are liable to come away from time to time and cause attacks of renal colic. Or it is possible for such fragments to become impacted in the ureters and cause fatal suppression of urine.

HÆMORRHAGIC PYELITIS.

Cases characterized by intermittent attacks of pain over one kidney and bloody urine, or of bloody urine alone, are of not uncommon occurrence. Only a moderate number of these cases have found their way into print, but conversation with physicians and surgeons shows the existence of many unreported cases. The symptoms may follow either a mild or a severe course.

The milder form of the disease seems to be of most common occurrence in young girls. The only marked symptom is the appearance in the urine for days or weeks of red and white blood-cells and of epithelium from the pelvis of the kidney. The patients may also suffer from hysteria or from disturbances of digestion. After one or more of such attacks the patients recover altogether. No treatment is necessary, except to relieve disturbances of digestion and improve the general health.

The severe form of the disease is seen both in males and females. From time to time the patients suffer from attacks of pain referred to one kidney, and the urine contains large quantities of blood. With the cystoscope the blood can be seen to come from the ureter belonging to the affected kidney. Some patients after a number of such attacks have no further trouble and recover entirely. In others, however, the pain is so intense or the loss of blood so threatening that surgical operations have been undertaken for their relief, usually with the expectation of finding calculi in the kidney. These kidneys have been simply cut down on and felt of, or needled, or split open, or extirpated. It is a curious fact that recoveries are reported after each one of these different operative procedures.[1]

In the only one of these kidneys which I have had the opportunity of examining after extirpation the kidney-tissue itself was normal and contained no extravasated blood. The mucous membrane of the pelvis and calyces was much thickened, its stroma infiltrated with cells, and the epithelium irregular.

Treatment.—The stricture of the urethra, enlarged prostate, cystitis, or other cause of the pyelitis is to be removed as entirely as is possible. For the chronic pyelitis the improvement of the general health, an out-of-door life in a suitable climate, and the use of such waters as those of Ems and Poland

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[1] Senator, *Berl. klin. Wochensch.*, Jan., 1891; Sabatir, *Rev. de Chir.*, 1888; Schede, *Jahr. d. Hamb. Stadtkrankenhaus*, 1889.

Springs are often of great service. A considerable number of drugs have been employed for the cure of the pyelitis—the mineral acids, the tincture of the chloride of iron, the alkalies, the vegetable astringents, salol, and others. In pyelitis in the female good results are reported by Bozeman from opening the bladder and washing out the pelvis of the kidney through the ureter. If the distention of the pelvis with purulent fluid be very great, or if the kidney itself undergoes suppuration, the kidney must either be opened or removed. The exact determination of the existence of a pyelitis can be made with a cystoscope if pus can be seen to issue from one or both ureters.

RENAL CALCULI.

The solid constituents of the urine may be precipitated in the pelves of the kidneys in the form of sand, gravel, or calculi. The calculi are formed of uric acid, of uric acid with a shell of oxalate of lime, of oxalate of lime, or of the phosphates. Calculi composed of cystin are of a light-yellow color and lustrous, looking something like beeswax; they are of rare occurrence. Calculi composed of xanthin, of fatty or saponaceous matters, of carbonate of lime, and of fibrin have been described in rare cases.

Etiology.—Renal calculi are found in persons of all ages; they are of more frequent occurrence in males than in females. A sedentary life, gout, any conditions which produce an exaggerated excretion of uric acid, of oxalate of lime, of phosphates, or of cystin, and inflammation of the mucous membrane of the pelves of the kidneys, may act as causes.

Symptoms.—Small calculi may be formed in the pelves of the kidneys, be passed through the ureters into the bladder, and finally escape through the urethra without giving any trouble.

Larger calculi, which are formed in the pelves and pass through the ureters, cause by their passage attacks of renal colic. During an attack of renal colic the patients have severe pain, coming on suddenly or gradually, referred to the kidney and ureter, and radiating down to the groin, or simply a diffuse abdominal pain. The testicle is retracted, sometimes painful and swollen. Occasionally the pain is so severe that the patients faint or have general convulsions. Vomiting or retching is sometimes frequently repeated and very distressing. There may be a moderate rise of temperature.

The urine, during the attack, is passed frequently, in small quantities, often with a good deal of pain, and may contain blood. After the attack a considerable quantity of urine is passed, which for a time may be bloody.

Most attacks of renal colic last only for a few hours, but they may be protracted, with intermissions, for a number of days. The same patient may have one attack or several attacks.

The calculus, instead of getting through the ureter into the bladder, may become impacted. If the patient has only one kidney or if the ureters of both kidneys are obstructed, the urine is entirely suppressed, and the patients die after a number of days in the condition of chronic uræmia. If the ureter of one kidney is obstructed, either hydronephrosis or pyonephrosis follows.

The calculi, after being formed in the pelvis of the kidney, may remain there. When this happens the patient may never have any symptoms, nor any considerable change in the kidney; or without any symptoms the kidney may become atrophied. More frequently the patients have repeated attacks of pain over one kidney and bloody urine. After a number of attacks they may finally pass a calculus. If the attacks cease and no calculus is passed, we cannot tell whether the patient has a renal calculus or the form of pyelitis with pain and bleeding which I have already described.

Very often the presence of the calculus in the pelvis of the kidney is accompanied with inflammatory changes in the pelvis or in the kidney. The calculus may cause the inflammation, or a chronic pyelitis may be followed by the formation of phosphatic concretions.

If there be chronic pyelo-nephritis, the patients have pain and tenderness over the diseased kidney, either continuous or in attacks. The urine at intervals contains pus and blood, sometimes fragments of the calculi. The dilatation of the pelvis of the kidney may form a tumor of appreciable size. The patients lose flesh and strength, they have an irregular fever, they are liable to have chronic nephritis of the other kidney or waxy degeneration of the viscera.

In some cases the calculus causes but little change in the pelvis of the kidney, but the kidney itself becomes the seat of chronic diffuse nephritis, with a little exudation. The urine then becomes of low specific gravity and contains a little albumin. The patients are liable to attacks of contraction of the arteries, with dyspnœa or cerebral symptoms, or they may become somewhat dropsical.

Treatment.—To prevent the formation of uric-acid and oxalate-of-lime calculi we regulate the diet by excluding as far as possible the starches and sugars. We insist on sufficient exercise in the open air. The patients should spend several weeks every year at one of the natural alkaline springs, or, if this is not possible, should take from time to time one of the alkalies largely diluted with water.

The treatment of the attacks of renal colic is directed to the relief of the pain and to hastening the passage of the calculus. The means usually employed for this purpose are hypodermic injections of morphine, inhalations of ether or chloroform, and the hot bath.

A calculus impacted in the ureter or one in the pelvis of the kidney can be removed only by surgical means.

THE CYSTIC KIDNEY.

Cysts are formed in the kidney both during intra-uterine and extra-uterine life.

The congenital cystic kidney is a very remarkable pathological condition. There is enormous enlargement of either one or both kidneys with the conversion of the tissue of the organ into a mass of cysts. The cysts are of all sizes, and are separated from each other by fibrous septa or compressed kidney tissue. They contain a clear, yellow, acid fluid, holding in solution the urinary salts, or the fluid is turbid and brown, and contains blood, uric-acid crystals, and cholesterin. The cysts are lined with a single layer of flat epithelial cells. They seem to be formed by a dilatation of the tubules and of the capsules of the Malpighian bodies. As causes for such a dilatation are found obliteration of the tubes in the papillæ, or stenosis of the pelvis, ureters, bladder, or urethra. Other congenital malformations are often associated with this one.

In adult life we find three varieties of cystic kidney:

1. In kidneys which are otherwise normal there are one or more cysts filled with clear or brown serum or colloid matter. These cysts do not appear to interfere at all with the functions of the kidneys.

2. In chronic diffuse nephritis, especially in the atrophic form, groups of tubes are dilated. Apparently there is obstruction of one or more of the larger tubes in the pyramids, and this causes dilatation of a corresponding group of tubes. Such a dilatation may be moderate in size, or it may form cysts visible to the naked eye.

3. Both kidneys are very much enlarged, and converted into a mass of cysts containing clear or colored serum or colloid matter. The nature of these cysts is uncertain. It is possible that they are congenital. They are sometimes associated with similar cysts in the liver. Their clinical history resembles that of some of the cases of chronic diffuse nephritis without exudation. The patients go on for a long time without symptoms, except that the specific gravity of the urine gradually falls and a little albumin is occasionally present. Finally they have an attack of general convulsions or of coma, and die in a short time.

New Growths of the Kidney.

The most important new growths of the kidney are those which belong to the classes of sarcoma and adenoma.

The Sarcomata grow from the kidney itself or from its pelvis. They are composed of connective tissue with an excess of cells, with which may be mixed mucous tissue or muscular tissue. These tumors often reach a large size, and may grow for a number of years before they cause death. They form a hard abdominal tumor, which at first retains the position and shape of the kidney, but may finally become so large as to occupy a considerable part of the abdominal cavity.

They are found as congenital tumors, are rather frequent in infants and children, and are occasionally met with in adults.

The Adenomata grow in the cortex of the kidney in the form of nodular tumors. They may follow the papillary or the tubular type. In some cases the tumor or tumors never attain any considerable size, are not malignant, and give rise to no symptoms. In other cases the tumors become much larger, and may then behave like malignant growths. These large tumors are very vascular. The adenomata which run a malignant course with the formation of metastatic tumors are often called carcinomata.

Symptoms.—The sarcomata and adenomata, so far as their symptoms are concerned, may conveniently be described together. In both of them there are four principal symptoms: a tumor, pain, hæmaturia, and loss of nutrition.

The tumor is appreciable as soon as it has reached a sufficient size. While it retains the natural position and outlines of the kidney the diagnosis is comparatively easy, but as the tumor becomes larger and adhesions are formed it becomes more difficult to distinguish it from other abdominal tumors.

Hæmaturia is present at some time in the disease in about half the cases. In some cases the hæmorrhage is excessive, and is followed by rapid anæmia and exhaustion; more frequently the loss of blood is moderate, and sometimes only to be discovered under the microscope; it is apt to recur at intervals of days or weeks.

The pain is referred to the diseased kidney. It is by no means a constant symptom, but yet may occur early and be throughout a prominent feature. It is apt to come on in attacks, and to radiate downward along the course of the ureter.

Loss of appetite, nausea, and vomiting are troublesome symptoms in some of the cases.

The loss of flesh and strength surely comes sooner or later, but it is curious in some cases how long the general health may remain unaffected, and how long life can be prolonged even with enormous tumors.

Treatment.—The same rule seems to hold good for these tumors in the kidney as for the same tumors in other parts of the body. If the kidney be removed while the growth is still small, the prognosis is fairly good. If it be not removed until the tumor is large or until metastatic tumors have been formed, the prognosis is bad.

DISEASES OF THE MOUTH AND TONGUE.

BY WILLIAM PEPPER.

DISEASES OF THE MOUTH.

STOMATITIS.

THE term "stomatitis" is a general one, indicating the various kinds of inflammation of the mucous membrane of the mouth, including the lips, gums, cheeks, and palate. When but one of these structures is involved some other term than stomatitis is better employed.

CATARRHAL STOMATITIS.

Definition.—A simple superficial inflammation of the mucous membrane of the buccal cavity not attended by ulceration.

SYNONYMS.—Erythematous stomatitis; Acute stomatitis; Simple stomatitis; Pultaceous stomatitis.

Etiology.—There is a distinctly greater tendency to the affection in infants than in older subjects. The causes of the disease are various and often obscure. They may be either primary or secondary, depending on some general or more remote local disorder. Among the diverse local exciting causes are food which is too hot or too cold; certain drugs, as preparations of arsenic, iodine, bromine, mercury, and lead; corrosive substances; excessive use of tobacco or spirits; the pus from an alveolar abscess or mechanical irritation, as from broken or carious teeth; in the case of sucklings, prolonged sucking at an empty breast or an imperfectly developed or an artificial nipple; lack of cleanliness about the mouth; the presence of hair lip, or cleft palate. Normal dentition does not produce any disorder of the mouth. When, however, dentition assumes pathological features, it is not rarely attended by catarrhal stomatitis.

Exposure to cold must be recognized as an occasional cause. Secondary catarrhal stomatitis may depend on gastric or intestinal derangements, affections of the tonsils and pharynx, coryza, various febrile diseases, especially scarlet fever and measles, and occasionally on lactation, and on menstruation or pregnancy.

Pathological Anatomy.—Circumscribed inflammation of the mucous membrane is more frequent than the diffuse form. The lesions found may be conveniently divided into two varieties. In the first, which is simply an erythematous process, there is bright redness and dimished secretion of the mucous membrane, which is dry and glistening. But slight swelling occurs. In the second, the true catarrhal process, there is hypersecretion with decided swelling and increased heat in the mouth. The mucous membrane is generally red, but may be whitish from the accumulation of desquamating epithelium. The swollen lining of the cheeks is often marked by the imprint of the teeth. The lips are apt to be swollen and tense, and in bad cases the blood-vessels, particularly over the hard palate, are injected. The mucous follicles may be distended and prominent. Slight abrasions or mere superficial ulcerations may occur as a complication in bad cases. The tongue is somewhat swollen, and indented by the teeth. Its coating is white, and at first dry, but later moist. In stomatitis associated with fever, hypersecretion may not occur at all, or it may finally give place to dryness of the mouth. The tongue then becomes red, dry, and fissured, and the rest of the oral mucous membrane undergoes a somewhat similar change.

With stomatitis is associated engorgement of the neighboring lymphatic glands corresponding to the degree of the oral inflammation.

Symptomatology.—The symptoms vary with the severity of the attack. Apart from the appearances already described, the principal evidence of the disease is pain. In the infant this is shown by fretfulness and crying. The child makes an effort to nurse, but quickly drops the nipple. In older children and in adults there is pain on mastication and in deglutition. Taste is early affected. The secretions of the mouth, at first diminished, may soon be greatly increased, and often become viscid and ropy. In infants there is usually excessive dribbling of saliva. The breath has an unpleasant odor, sometimes sweetish and sometimes sour. The appetite is not affected in cases of moderate severity. More or less fever is generally present.

Complications.—The complications of stomatitis are few. Diarrhœa occasionally attends it in children, as does dyspepsia. Vomiting may follow strenuous efforts to loosen and expectorate the viscid secretion. Convulsions in children may develop, perhaps the result of reflex irritation from the mouth. The submaxillary glands swell in severe cases. Eczema of the chin may be set up by the overflowing saliva. Sometimes severe catarrhal stomatitis is complicated by the development of a single or a few superficial ulcers, which heal as soon as the stomatitis is relieved.

Diagnosis.—The disorder can scarcely be mistaken for any other affection. It is only important to determine whether the stomatitis is a primary or a secondary one.

Prognosis and Course.—The prognosis is nearly always favorable. Only in weakly infants is it sometimes serious, owing to the interference caused with the ingestion of food. There is no fixed limit to the duration of the disease, since this will depend upon the removal of the cause, but it seldom lasts over a week. Weakly children who have once suffered from it exhibit a great tendency to repeated attacks upon the slightest irritation, so that the affection soon becomes chronic in this way.

Treatment.—Prophylactic treatment is of great importance. In the infant the interior of the mouth should be cleansed regularly but gently, preferably with absorbent cotton. The nipples, whether natural or artificial, should be kept scrupulously clean. In older subjects the use of the tooth-brush and quill must not be neglected. In the course of prolonged febrile affections particular attention must be given to keeping the mouth frequently moistened with cool water.

The treatment of the attack itself consists in the removal of the cause whenever this can be discovered, and in the use of local applications. Food should be warm or, oftener, cool, according to the sensations of the patient. The mouth should be washed frequently and carefully with solutions of boric acid (1–3 per cent.), borate of sodium (5 per cent.), bicarbonate of sodium (5 per cent.), sulphate of zinc (0.5 per cent.), tannic acid (2–5 per cent.), salicylic acid (0.5 per cent.), barley-water, slippery-elm water, gum-water, or even simple cool or tepid water. These solutions may be conveniently applied in the form of a spray. One of the best applications in obstinate cases, or where ulceration has taken place, is nitrate of silver in ½ per cent. solution. It should be preceded by thorough cleansing of the mouth. For the relief of pain ice may be sucked or a very dilute solution of cocaine be applied. The cocaine, however, should not be used in children who cannot be taught to expectorate the solution and cleanse the mouth afterward. Attention to the state of digestion is usually required. If a laxative be indicated, it should be in the form of small doses of a mild saline. If the stomach be irritable, minute doses of silver nitrate are valuable. Muriatic acid in small doses, freely diluted, with or without a mild vegetable bitter, will be found to hasten convalescence.

THRUSH.

Definition.—A disease of the mucous membrane of the mouth characterized by the development in it of a certain fungus and attended by slight inflammatory changes.

SYNONYMS.—Parasitic stomatitis; Mycotic stomatitis; Soor (German); Muguet (French); Stomatomycosis.

Etiology.—The fungus producing the affection is the primary cause, and without it the disease would not exist. There are, however, certain predisposing causes. The affection is by far more common during the first weeks of life, though it may occur in older children or adults the subject of debilitated or cachectic states. This is not the result of any predilection of the disease for any age, but rather of the presence of conditions favoring its development. Possibly one of these is the greater degree of rest of the mouth and tongue present in early infancy and in debilitated subjects, which offers the micro-organisms a greater opportunity to find a foothold and to grow. Thrush is more frequent in hot weather, on account of the greater ease with which the fungus develops at this time. The disease is evidently contagious, and is consequently far more frequent in hospitals and homes for infants. The exact nature and source of the contagion and the various ways in which it may be diffused are not fully known. It is certainly transmitted from one child to another by the mother's nipple or by an unclean nursing-bottle. It has been claimed also that a child may be infected during birth, by thrush affecting the vulva of the mother. This, however, must be very unusual.

To what degree the spores of the fungus permeate the air is not known, though it seems probable that in infected children's wards the air is charged with them. A deterioration of the general health seems less a predisposing factor, *per se*, than does an unhealthy state of the buccal mucous membrane, since apparently perfectly healthy children may be attacked. At the same time, it is certain that a falling off in health of a baby may be attended by a condition of the mucous membrane which renders this a fit nidus for the growth of the fungus. Thus, any severe acute or chronic illness, improper feeding, imperfect hygienic surroundings, and the like may act in this manner. The most frequent condition which thus predisposes to thrush is gastro-intestinal disorder, and the association between the two conditions is often very intimate. It is not certain, however, whether the gastro-intestinal disturbance has any peculiar causative power.

It may be definitely asserted that any form of stomatitis renders the lodgment and growth of the fungus possible; but the most common predisposing alteration of the mucous membrane is a catarrhal stomatitis, even of the mildest grade.

It is very commonly claimed that an acid state of the secretion of the mouth is essential to the growth of the fungus. Experience indicates, however, that it will grow in neutral, or even slightly alkaline media. The saliva in this disease is, it is true, acid, but probably oftener as a result of the presence of the fungus.

Pathology and Pathological Anatomy.—The exact nature of the organism—which provisionally may be called saccharomyces albicans—has not yet been determined. It was first described by Berg in 1843. It was soon after again described by Robin under the title of Oïdium albicans; Hallier called it Oïdium lactis. Grawitz claimed that the fungus was in reality the mycoderma vini, but about the same date Reess denied the truth of this, and described it as saccharomyces albicans. Plaut believed it to be Monilia candida. It has also been maintained that the fungus is a mixed form. It is decidedly polymorphic, the form depending upon the nature of the media upon which it is cultivated. Roux and Linossier have shown that there is a yeast form made up principally of cells as seen in the yeast fungus. There is also a form which is very largely filamentous. Under certain conditions the morphology becomes still more complex. In the mouth the fungus produces white patches resembling curdled milk in color, and varying from a pin-point size upward. The spots may be few, or very numerous, discrete or coalescing to a great extent. They are covered by epithelium and slightly elevated, and can be removed only with the exercise of considerable force. This leaves a raw surface which bleeds easily. In the later stages the patches become duller in color and are more easily removed. Examined more minutely, it is found that the parasite first develops and spreads in the form of spores between the layers of epithelial cells. Mycelial threads now grow outward and, especially, inward toward the connective tissue. The mucous membrane in the neighborhood of the patches is smooth and redder than normal. There is some inflammatory production of new cells, but without pus. Some degree of general catarrhal stomatitis is usually to be observed.

The spots occupy principally the dorsum and edges of the tongue, but the hard palate and the inner surface of the cheeks and lips are also favorite seats. Other parts of the mouth may be involved, and the growth may spread to the fauces and pharynx, and even to the gastro-intestinal tract. It has indeed been found in various internal portions of the body, including the liver, lungs, brain, and blood-vessels.

Symptomatology.—The symptoms vary greatly, and depend rather upon the attending stomatitis than upon the mere presence of the parasite. In infants there may be witnessed the same indisposition to nurse which was described as a symptom of catarrhal stomatitis. Swallowing is painful if the disease extend to the fauces, and it may be impossible if the œsophagus becomes involved, owing to the blocking of its lumen by the mould. Such symptoms as wasting, debility, and the like are referable to the disease with which the oral affection is attended. There is an exception, however, in the case of disturbances of the gastro-enteric tract, which are often apparently due to the spread of the fungus to this region, or more probably to the swallowing of the mould and the consequent irritation set up by its chemical products.

Complications.—A rather chronic ulcerative process may occasionally remain after the detachment of the fungus from some of the patches. Gastro-intestinal catarrh is a very frequent complication, as already stated. Redness and excoriation of the parts around the anus and the genitals, so often present in thrush, is the result of a complicating diarrhœa.

Diagnosis.—At the outset the patches on the tongue look greatly like curdled milk. The latter, however, can at once be easily removed by the finger. Thrush can scarcely be confounded with any other disease, and if there be any doubt a microscopic examination will settle the matter.

Prognosis and Course.—Thrush occurring in a healthy child and appropriately treated offers an entirely favorable prognosis. In a few days to a week it should be well, having injured the patient little if at all. In greatly debilitated children it is apt to be indefinitely prolonged until death comes as a result of the general condition, except in so far as pain has interfered with the ingestion of food. The gravity of the prognosis is in proportion to the extent of the eruption and the youth of the subject.

Treatment.—Prophylaxis is of the utmost importance. As far as possible, the entrance of the spores must be prevented by the strictest cleanliness of everything which is to be put in the child's mouth. The free use of boiling water constitutes the best means for this end. The mucous membrane of the mouth also must be maintained in a clean and healthy condition by frequent washing with weak alkaline solutions.

If the disease has already commenced, the chief attention is to be directed to removing the fungus and preventing its return. The patches must be rubbed away by the finger, covered with a soft wet cloth, in as gentle a manner as possible, and this procedure repeated every two or three hours; the cloth may be moistened with an alkaline solution, since this softens the epithelial covering. After the washing the diseased parts must be sprayed or painted with the remedy to be employed for the destruction of the fungus. For this purpose we may use solutions of borax (20 grs. to the ounce), hyposulphite of sodium (20 grs. to the ounce), boric acid, salol, salicylate of sodium (20 grs. to the ounce); permanganate of potash, bicarbonate of sodium (30 grs. to the ounce), and peroxide of hydrogen. Borax and honey is a remedy much employed. It is better, however, to dissolve the borax in glycerin, since saccharine substances may favor the development of the disease.

Such constitutional treatment must be given as is indicated by the general health of the patient or by the nature of any other malady present. It may be necessary to diminish the amount of sugar and starch in the food, in order to avoid fermentation and increase of acidity, which furnish conditions favorable to the development of the micro-organisms. Such dietetic treatment, combined with the employment of alkaline washes for the mouth, will effectually prevent the increase of acidity of the secretions.

APHTHOUS STOMATITIS.

Definition.—An inflammatory affection of the oral mucous membrane characterized by the formation of small scattered yellowish-white or grayish-white patches or very superficial ulcers.

SYNONYMS.—Aphthæ; Canker; Vesicular stomatitis; Follicular stomatitis; Herpetic stomatitis.

Etiology.—The disease is much more common in children up to the age of the second dentition. Imperfect hygiene of any sort, with its debilitating influences, seems to predispose to the affection. Constitutional debility or that resulting from overwork, pregnancy, lactation, or severe prolonged illness acts in the same manner. Such acute diseases as measles, malaria, acute gastro-intestinal catarrh, pneumonia, and scarlet fever are sometimes accompanied by aphthæ. Toward the close of wasting diseases, such as phthisis or cancer, and especially it has seemed to me when the abdominal organs are involved, aphthæ are apt to appear, and to prove intractable. Often, however, the affection develops in persons apparently in perfect health. The season of the year has been claimed to exert some influence in its production, since summer and autumn seem to produce the greatest number of cases. Excessive humidity of the air has been supposed to produce it, since inundations have sometimes been followed by epidemics of the affection. Various local irritations have been assigned as causes. Prominent among them is teething, but whether the association is causal or merely accidental is difficult to determine.

Pathology and Pathological Anatomy.—The exact nature of the affection has not been accurately determined. Bacteriological studies have not shown the presence of any characteristic micro-organism. Arguments have been advanced in favor of the infectious nature of the malady, and the disease has also been spoken of as identical with the foot-and-mouth disease of cattle, but there seems to be no sufficient ground for this theory. It has been claimed that the ulceration is the result of inflammation of the mucous follicles. The counter-claim has likewise been made that this is impossible, since the disease appears in parts of the mouth where there are no such follicles. Some writers state that the lesion is a solid exudation in the mucous membrane just below the epithelium, while others claim that the initial stage is the formation of a vesicle. From this latter point of view aphthæ is of nervous origin and closely allied to herpes facialis. The difficulty of detecting the vesicles in the mouth is readily explained by the differences in the conditions there and on the face respectively. Strictly speaking, the lesion is not an ulcer, since no pus is produced. This has been urged strongly by Bohn. The lesions may develop in any part of the mouth, although the most common situation is the inner surface of the lower lip. They may extend into the pharynx, although this is rare. The patches of aphthæ vary in size from that of a pin-head to that of a split pea, and are for the most part discrete, though two or more closely adjacent spots may fuse. In some cases, however, the disease assumes a confluent type. This form has been described as occurring particularly during pregnancy or epidemically after inundations, but is not confined to these conditions. The aphthous lesion, as first observed, consists of a small elevation of the epithelium with a red margin and a whitish centre. It can be barely perceived by the finger. There is present hyperæmia and cellular proliferation. In about twenty-four hours the papule loses its epithelial covering, and a small oval whitish or creamy patch is left, surrounded by a red margin. The ulcer, as it is called, is level with the surface, though its swollen edges may make it appear slightly excavated. In a few days the mass forming the central part of the patch has been encroached upon and lifted up above the surface of the surrounding mucous membrane by the newly-formed epithelial layer, and disappears; or the floor of the ulcer has gradually receded, a clean-cut sore uncovered by epithelium has been left, and this has gradually been covered by a new epithelial coating spreading from the periphery. No scar remains.

Symptomatology.—A certain degree of catarrhal stomatitis always precedes the development of the aphthous patches. Sometimes, too, the appearance of these is preceded by such constitutional symptoms as feverishness, loss of appetite, and evidences of gastro-intestinal disturbance. This, however, is unusual except in severe cases, and it is questionable whether such symptoms are at all connected with the oral affection. The amount of systemic disturbance varies a great deal. Restlessness, loss of appetite, and fretfulness may appear in infants, together with a moderately increased secretion of saliva. Slight fever is common. In the confluent type the constitutional symptoms may be severe. Vomiting and diarrhœa may be present, perhaps due to the extension of the process to the stomach and intestines, and the typhoid state may be developed. Pain is usually the most prominent symptom, since the spots are extremely tender to the touch. It may even exist to such a degree that nourishment in the case of infants is greatly interfered with. When aphthæ appear in the later stages in wasting diseases they may add to the suffering, and hasten the fatal result by their interference with taking food.

Complications.—The preceding catarrhal stomatitis is rather a symptom of the disease than a complication. Rarely ulcerous stomatitis is added as a complication. Herpes of the lips sometimes occurs. Whether gastro-intestinal catarrh is a cause or a complication is undecided.

Diagnosis.—In the discrete form the diagnosis is easy. The presence of redness and swelling of the mucous membrane of the gums at their junction with the lips arouses the suspicion of a concealed aphthous ulcer. The confluent form may sometimes resemble diphtheritic inflammation, but close inspection or a day of waiting will probably discover the difference, as isolated aphthous ulcers are apt to appear here and there. Aphthæ may simulate ulcerous stomatitis. The latter is to be distinguished by the foul breath and the tendency to hæmorrhage and to breaking down of the mucous membrane, besides its primary situation at the junction of the teeth and gums.

Prognosis and Course.—The prognosis in the discrete form is entirely good. The disease seldom lasts longer than ten days at the outside, and generally not more than from four to seven days. In greatly debilitated subjects the affection may become almost chronic, owing to repeated relapses. Some persons appear to be particularly prone to the occurrence of the disease. The cure of the confluent form is more difficult, depending upon the nature of the primary disorder. This variety often lasts a couple of weeks.

Treatment.—As in catarrhal stomatitis, it is well to commence treatment with a mild saline laxative. Any gastric or intestinal derangement should be appropriately treated. Locally, the same care for cleanliness should be employed as was recommended for catarrhal stomatitis. Ice or cold water or demulcent lotions give relief. Distinct benefit can be obtained from the application of strong solutions of nitrate of silver. Sulphate of copper may be employed in the same manner. These caustics should be applied with care, or the ulceration produced may be more entensive than the original lesion. Permanganate of potash in 1 per cent. solution, carefully painted upon the individual spots, has been highly praised by Baginski. A saturated solution of iodoform in ether has proved in my hands a valuable application in many cases. Excellent results may be obtained from the combination of borax and chlorate of potash in a powder, of which a minute portion is placed in the mouth frequently during the day; or either drug may be administered in solution. Chlorate of potash may be given internally as well, since it is excreted by the saliva, and thus comes in constant contact with the ulcers. Other substances recommended are sulphate of zinc (20 grains to the ounce); boric acid (5 grains to the ounce); carbolic acid (5 grains to the ounce); bichloride of mercury (1 grain to the ounce); and astringent lotions of various sorts.

Solutions of cocaine may be employed, though with care, when the sensitiveness of the ulcers is very great. In the confluent form tonic and supporting treatment is sometimes indicated.

ULCEROUS STOMATITIS.

Definition.—An affection of the mouth characterized by extensive ulceration, situated principally upon the gums, but spreading by auto-infection to other parts of the oral mucous membrane.

SYNONYMS.—Ulcerative stomatitis; Ulcero-membranous stomatitis; Phlegmonous stomatitis; Stomacace; Putrid sore-mouth.

Etiology.—There are a number of predisposing factors. Sex is without much influence, but age is important. The disease is very unusual before the age of four years, and is most frequent between that of four and ten years. It is much rarer after this period, but may develop in adults, particularly among soldiers in camps and garrisons where it sometimes occurs epidemically. It is seen most frequently in the damp weather of spring and autumn. Improper feeding, insufficient ventilation, filth, imperfect clothing, and similar unfavorable hygienic conditions are powerful predisposing causes. General feebleness of constitution, as in rickets, syphilis, and tuberculosis, as well as the debilitated health following acute and chronic diseases, acts in the same manner. This is very true of the exanthemata, pertussis, pneumonia, and malaria. There is an individual propensity to the disease in some cases, so that an attack comes on after any slight illness or even in apparently perfect health.

Certain drugs are capable of producing it. This is true to some extent of lead, copper, and phosphorus, but particularly so of mercury. Whether or not contagion is an etiological factor has been a matter of dispute. The evidence is strong, however, in favor of the infectious nature of the disease. Besides the causes mentioned, which may be denominated general or systemic, a local cause is required, and consists in some disordered condition of the oral mucous membrane. This may be produced, perhaps, by a mechanical irritation, the presence of caries of the teeth or of tartar upon them, the excretion of mercury through the mouth, chronic hyperæmia, prolonged catarrhal stomatitis, or the action of some infection of unknown nature, since no specific microbe has been discovered. It is a curious fact that ulcerous stomatitis does not occur unless there are teeth. It would seem to indicate that neglect of cleanliness, allowing the presence of fermenting or putrefying particles in the interstices, may often serve as at least a predisposing cause.

Pathology.—The process always commences at the anterior aspect of the free border of the gums, generally of the lower jaw and upon one side, especially the left, according to Bohn. Thence it extends to the lips, cheeks, and edges of the tongue, but the dorsum of the tongue, the palate, and parts posterior to it escape. The first step is hyperæmia of the mucous membrane and swelling from the inflammatory exudation. Soon the gums become loosened from the teeth, and often somewhat everted and bleed easily. The upper free edge rapidly assumes the appearance of a narrow yellowish line, indicating ulceration, which rapidly broadens, and may involve the whole breadth of the gums. It may extend sufficiently in depth to invade the bone and cause its necrosis and loss of the teeth. The ulcers which may meantime have formed on the lips and cheeks often reach considerable dimensions. Their edges are not materially thickened, although the whole surrounding mucous membrane is commonly much swollen. The centre of the ulcer is covered with a soft grayish substance.

Histologically, the process is one of molecular necrosis, with abundant small-celled infiltration in the surrounding parts. Numerous bacteria are present, but none which can be called specific. The dead tissues show the forms of very few cells, but rather a granular detritus and some pus corpuscles. As recovery takes place, the yellowish detritus is removed and a new epithelial covering forms.

Symptomatology.—There are no general symptoms which precede the local ones. A very fetid or gangrenous odor to the breath and a profuse secretion of saliva are among the earliest symptoms, indicating the presence of the pathological lesions already described, and which inspection reveals. The saliva is commonly tinged with blood. The tongue is swollen and the lining of the cheeks is often puffy. There is pain upon taking nourishment, and young children may refuse food entirely. The general symptoms are usually slight, and there is only moderate fever. The child often becomes fretful, sleeps badly, and may lose flesh. In mercurial cases there is often a metallic taste in the mouth.

Complications.—Swelling of the submaxillary glands is so common as to be with equal propriety designated a symptom. Suppuration in them rarely takes place. An offensive diarrhœa may occur, possibly the result of swallowing the secretions from the ulcers. Nausea and vomiting may be produced in the same manner. Aphthæ may complicate the stomatitis. Gangrenous stomatitis may sometimes develop.

Diagnosis.—The disease can scarcely be mistaken for any other. Aphthæ upon the free border of the gums might be confounded with ulcerous stomatitis in the early stages, but the diagnosis could not be long in doubt. Moreover, aphthæ, if confluent, may spread to the pharynx. The patches, too, are more superficial. Noma is distinguished by extensive induration and tumefaction of the cheek and by the presence of blackened sloughing tissue.

Prognosis and Course.—Uninterfered with, the disease lasts for a variable time, perhaps months. When the cause is persistent, as in rickets and syphilis, the process may be indefinitely prolonged. It has a tendency to spread rather than to recover of itself. Sometimes it assumes a mildly chronic form from the outset. Under treatment the prognosis is excellent, and the disease disappears in a week or ten days, unless it has involved the bone. Recurrences of the affection are common. Death may occasionally occur in bad cases as the result of exhaustion, but it depends rather on a primary condition of malnutrition. Death may also result from the development of noma as a complication.

Treatment.—Prophylactic treatment consists in removing all possible predisposing and exciting causes by attending to the general health in every way, by avoiding possible infection by persons already attacked, and by keeping the oral mucous membrane in a healthy state. For the actual treatment of the disease almost a specific is found in chlorate of potash. This may be used in the form of lotions of the strength of from 15 or 20 grains to the ounce, while at the same time it should be given internally. The proper dose for a child of five years of age is about 3 grains every three or four hours. It may be administered in such a form as the following:

℞. Potassii chloratis, ʒj ;
Tr. cinchonæ comp.,
Syr. zingiberis,
Aquæ puræ, āā. f℥j.—M.
Sig. A teaspoonful in water every 3 or 4 hours.

Tincture of the chloride of iron may be advantageously combined with it for its tonic effect. Under the administration of chlorate of potash, improvement begins in twenty-four to forty-eight hours, as shown by the marked decrease in the salivation, and within a week all the symptoms will generally have disappeared. The treatment must, however, be continued for some time to guard against relapses. In more chronic cases chlorate of potash sometimes fails. Good results may then be had from the local employment of such agents as solutions of thymol, salycilic acid, carbolic acid, boric acid, permanganate of potash, and nitrate of silver. Probably the best of these are the last two. The former in the strength of 3 grains to the ounce may be applied frequently with a brush; the latter, in a solution of 20 or more grains to the ounce, can be painted carefully on only the affected parts several times a week. All carious teeth or portions of necrosed bone must be removed or ulceration may persist. Tonic treatment is of the utmost importance in order to prevent relapses.

MEMBRANOUS STOMATITIS.

Definition.—A form of inflammation of the mouth characterized by the formation of a pseudo-membrane upon the mucous surface which leaves an ulcer when removed.

SYNONYMS.—Diphtheritic stomatitis; Croupous stomatitis.

Etiology.—The disease is unusual, and owes its causation to one of several factors, the commonest of which is diphtheria. The lesion is then almost always secondary to diphtheritic inflammation elsewhere in the body, although I have occasionally noted primary diphtheria of the mouth, and similar cases have been recorded by others. Some writers describe a form as "croupous," secondary to a non-diphtheritic pseudo-membranous affection of the fauces. The term "croupous" is certainly a misnomer. Whether or not there is actually a difference between these forms is still uncertain. From the standpoint of pathological anatomy there does not appear to be any, except in so far as that a specific germ may produce the diphtheritic affection.

In the category of membranous stomatitis can also best be included cases which result from the action of caustic fluids upon the mouth.

Pathology.—In the diphtheritic form there is injection of the mucous membrane, and in a very short time a pseudo-membrane appears upon it. This remains several days and then separates, leaving a superficially ulcerated surface. The pseudo-membrane may become detached in mass or may slowly disintegrate. It consists of a fibrinous exudation with leucocytes, which is situated in the mucosa, and which has undergone a coagulation-necrosis. It may involve only the superficial epithelial layers, but if more deeply situated, it will seem rather within the mucous membrane, can only be removed with difficulty, and leaves a bleeding surface behind it. When thus artificially removed, it is apt to re-form. The most frequent situation of secondary diphtheritic stomatitis is the tongue, and after this the cheeks, lips, and gums. In primary diphtheritic stomatitis the lips are generally first attacked, and after this the other parts of the mouth. The characteristic micro-organisms of diphtheria are of course present.

In those cases due to the action of hot or caustic liquids there is a sudden production of a pseudo-membrane by the coagulation and consequent killing of the surface layers, whence the necrosis extends inward. Other cases of membranous stomatitis have been described in which the method of origin was entirely obscure. Such, for instance, is the case reported by Bax, in which the attacks of this condition recurred at intervals during twenty-five years.

Symptomatology.—Some degree of catarrhal stomatitis is a frequent accompaniment of diphtheritic stomatitis. Salivation is nearly always present. The odor of the breath is fetid. Slight or even severe hæmorrhage may take place. There are decided swelling and tenderness of the lymphatic glands below the jaw. The appearances in the mouth have already been described.

Diagnosis.—The disease must be distinguished from confluent aphthæ and from ulcerous stomatitis. The diagnostic symptoms have already been described in considering these two affections.

Prognosis.—In membranous stomatitis secondary to diphtheria of the pharynx, the affection of the mouth is an indication of the intensity of the process, and the prognosis is very grave. In the primary diphtheritic form the prognosis is less grave. In those cases dependent upon local irritants it is, as a rule, and as regards the lesion of the mouth merely, favorable, unless the destruction has been very extreme.

Treatment.—The treatment of the diphtheritic variety is that of diphtheria of the pharynx. The removal of the pseudo-membrane and the application of some of the numerous agents advised for use in the throat are to be recommended where the patient seems to be suffering more from the local process than from general infection.

In forms not dependent upon a specific virus it may be advisable to use astringent lotions for the ulceration remaining after the pseudo-membrane has been removed.

BEDNAR'S APHTHÆ.

Allied to ulcerous stomatitis in so far that ulceration is present, but widely differing from it in every other particular, is the affection known as Bednar's aphthæ.

Definition.—Superficial ulcers, generally one or two in number, which form upon the hard or soft palate in early infancy.

SYNONYM.—Placques pterygoïdiennes.

Etiology.—The disease is seen only up to about six weeks of age. Probably several different factors may act as the cause of the ulcers. Among them are the development of milium on the palate, followed later by ulceration and the occurrence of retention cysts terminating in the same way. The commonest cause of Bednar's aphthæ is, however, mechanical injury. This may be the result of the impingement of an artificial nipple upon the hard palate, or the mere act of suckling by a child who already has catarrhal stomatitis; but far more frequently it has been proved to be the result of rough washing of the mouth by the nurse's finger. Careful experiments made by at least two different investigators have proved that children in whom the toilet of the mouth was not practised rarely developed the disease.

Another, and evidently quite different, affection, from the point of view of prognosis at least, has been described by Parrot and others. In this the ulcers are situated as described, but occur only in children who are suffering from great inanition.

Pathology.—The first stage of the lesion consists in injection of the mucous membrane, which is soon followed by a grayish exudation, and then by the formation of superficial ulceration. The ulcers are situated on the hard palate or on that and the soft palate. In the cases described by Parrot the ulceration extended more deeply, and even involved the bone.

Symptomatology.—The only symptom produced is pain, shown by disinclination to nurse. Failure of nutrition may follow as a result of this.

Complications.—Thrush sometimes complicates the disorder. Gastro-intestinal disturbance may develop.

Diagnosis.—The disease cannot be confounded with any other affection. The suggestion of hereditary syphilis may occur, but is refuted by the antecedents and by the associated symptoms of the case.

Prognosis.—The prognosis is nearly always favorable, the tendency of the ulcers being to heal. If they last too long, the child may die from the interference with proper nourishment or from some complication. The prognosis of cases such as those described by Parrot is very unfavorable, the ulcers running a chronic course and rarely healing.

Treatment.—This is entirely negative. If the cause be removed, the ulcers will promptly heal. If they seem at all slow in doing this, they may be touched with a weak solution of nitrate of silver.

SIMPLE ULCERATION OF THE MOUTH.

The very distinctive appearance and course of the affections already described leave unconsidered some other forms of ulcers of the mouth. Among these is the development of a single or a few ulcers, sometimes consecutive to catarrhal stomatitis, sometimes the result of injury by a tooth and the like, and which have neither the seat nor the severity of those of ulcerous stomatitis, nor present the complex of symptoms of aphthæ. These ulcers are superficial and somewhat painful, and disappear readily when the cause is removed.

Perhaps best mentioned in the same connection is the fissuring at the angle of the mouth, with inflammatory thickening of the epithelium here. This resembles the fissuring of congenital syphilis. It gives rise to some pain. An epidemic of it was described by Lemaitre, and the disease was named "la perlèche," from the disposition of the patients to lick the painful spots.

GANGRENOUS STOMATITIS.

Definition.—An affection of the mouth characterized by gangrenous destruction of the gum and inner surface of the cheek, which spreads rapidly and is commonly fatal.

SYNONYMS.—Cancrum oris; Noma; Gangrene of the mouth.

Etiology.—The disease is a rare one, even in hospital practice. Certain factors exercise a powerfully predisposing influence. Decidedly more females are attacked than males. It has been observed in adults and in infants, but is much more frequent between the ages of two and five years. It appears to be more common in damp countries, such as Holland. More cases are met with in spring and autumn, but it is doubtful whether the season itself exerts any influence. Some previous deterioration of the health is a requisite etiological factor, as the disease does not occur in healthy subjects. Any improper hygienic surroundings may be sufficient to determine its occurrence. The previous existence of chronic or acute disease may bring on noma in a similar way. This is especially true of infectious diseases, but particularly of measles, since probably more than half the cases of gangrenous stomatitis are preceded by this disease. Malaria is a frequent predisposing cause in India. Local changes in the mucous membrane may act as a predisposing cause. Consequently ulcerous stomatitis is at times followed by noma, and aphthæ has also preceded it.

The question as to the infectious nature of the disease is not even yet determined. Cases arise spontaneously in which it seems impossible for the affection to have been imparted by contagion. On the other hand, several cases have sometimes developed in one family or in a hospital.

Pathology.—The disease almost always attacks both the cheek and the adjacent gums. Occasionally only the cheek or only the gums are affected. Very rarely the disease is bilateral. It is difficult to determine the exact mode and place of starting, owing to the great rapidity of development. It is at least certain that it is nearly always the mucous membrane which is first attacked, although some cases have been reported in which the disease began in the substance of the cheek. The process probably commences as a bleb upon the mucous membrane of the cheek or the junctions of it with the lips or gums.

This is rarely seen, since it speedily changes into an ulcer with a dark-grayish sloughing surface which is accompanied by diffuse swelling of the cheek. The ulceration immediately or after a day or so becomes gangrenous, is covered with a brownish eschar, and spreads rapidly in all directions, including that of depth. The cheek soon presents a well-defined indurated nodule in its substance, and the skin over this becomes tense and red, then livid, and finally blackish. The discoloration and induration spread, a bleb often forms externally over the position of deepest ulceration within the mouth, and the skin of the cheek finally breaks. At last this gangrenous spot meets the similar area upon the inside, and the perforation is complete. The process now extends and eats away the cheek, while meantime the changes within the mouth often extend rapidly. The teeth may be loosened, the bones become necrotic and portions of them drop out; the tongue, lips, palate, eye, nose, and even the ear, may be eventually involved. The rapidity of the changes described varies considerably. Perforation may take place in twenty-four hours, but oftener after three to four days. It may, however, not occur for two weeks. In rare instances I have seen the gangrenous process terminate favorably without perforation of the cheek.

The process is that of rapid gangrene. A plugging by thrombi of the vessels in the surrounding infiltrated area prevents the occurrence of hæmorrhage.

There has as yet been no micro-organism found which can be considered certainly characteristic of the disease. Lingard has described a thread-like bacillus, and Ranke a peculiar streptococcus. Sansom described certain highly refractory bodies in the blood in noma, but admitted that they were not always present. Post-mortem lesions are found in various organs. A diffuse bronchopneumonia is very frequent. Membranous patches in the colon are quite common.

Symptomatology.—The symptoms vary very much, and are often at first masked by those of the disease that precedes the noma. Often the first evidence is the gangrenous odor which proceeds from the mouth, and which finally becomes unbearable. There is observed at the same time a diffuse swelling of the cheek, and inspection of the interior of the mouth will show that the affection has made considerable progress. There is not much pain. The general condition of the patient seems often to be very little affected at the outset or even after the process is well under way. Eventually, however, as the gangrene progresses, there ensues great depression of strength and delirium or insomnia may develop. There is moderate fever, which is apt later to take on a septic type. The pulse is weak, and finally becomes rapid and feeble. Appetite may be well preserved nearly to the end. The secretion of saliva is increased; the submaxillary lymphatic glands are little if at all enlarged; œdema of the feet is common. Finally the patient becomes apathetic, and dies in collapse or from exhaustion.

Complications.—Diarrhœa is so frequent that it is questionable whether it should not be called a symptom. It is probably due to the swallowing of gangrenous matter or to a complicating colitis. Vomiting is uncommon. Broncho-pneumonia is a very frequent complication. Gangrene of the lungs, palate, œsophagus, anus, genitals, or extremities may occur. Hæmorrhage is rare, but has occasionally caused death. Diphtheria sometimes complicates the affection.

Diagnosis.—When well established noma cannot be mistaken for any other disease. All the evidences of gangrene are present. Anthrax is distinguished by the fact that it commences on the exterior. Ulcerous stomatitis, if very severe, may sometimes cause necrosis of the bone and considerable destruction of the mucous membrane. It does not, however, produce a gangrenous odor; the ulcer is not covered by a brownish eschar, nor does it advance to destruction of the skin. As already stated, ulcerous stomatitis may sometimes take on a gangrenous change.

Prognosis and Course.—The prognosis is most unfavorable, though not invariably so. At least three-quarters of all cases die. The more extensive the lesion, the more fatal the disease. The chances are best when the lesion is confined to the gums. The presence of any complication adds to the gravity of the case. Recovery may take place before the disease has passed beyond the mucous membrane, but oftener it is only with the attendance of great deformity of the cheek. The duration of gangrenous stomatitis is one to two weeks or longer. Relapses very rarely occur.

Treatment.—Little can be done in the way of prophylactic treatment, since the disease comes so suddenly and without warning. A child in a hospital if attacked by the disease should be isolated, in view of the possible contagiousness. The treatment of the disease itself must be energetic to be of any avail. Prompt and thorough cauterization under anæsthesia with the actual cautery or with nitric acid or some other powerful caustic should be employed. Carbolic acid, Vienna paste, bromine, sulphate of copper, chloride of zinc, and other substances have been employed for this purpose. The applications should be made as early in the disease as possible. The effort must be made to penetrate into the tissue which seems still entirely healthy, while great care is used to prevent unnecessary injury to adjacent healthy parts. How often cauterization must be repeated depends upon the condition in each individual case. The after-dressing depends, too, upon the individual circumstances, but antiseptic dressings of some sort are always indicated. Iodoform in vaseline may be applied over the inner surface of the cheek, and externally the following may be smeared freely over the side of the face and neck:

℞. Iodoformi,	gr. xl;
Icthyol,	ʒj;
Lanolin,	ʒij;
Ung. petrolei,	q. s. ad ℥j.—M.
Ft. ung.	

All care possible must be taken to prevent the entrance of septic matter into the lungs and stomach, in order to avoid the development of diarrhœa and broncho-pneumonia.

The general health of the patient must be sustained in every possible way. Food should be administered in concentrated form and at frequent intervals, and nutrient enemata are sometimes necessary. Diarrhœa should be restrained, if possible, by small doses of opium by the rectum. Alcohol is well borne and should be freely given. The internal remedies which have seemed to be most valuable have been oil of turpentine in emulsion with lime-water, and dilute muriatic acid in solution with quinine and strychnine.

LEUKOPLASIA ORIS.

Definition.—A chronic affection of certain parts of the mucous membrane of the mouth or tongue characterized by the development of whitish or yellowish somewhat elevated and indurated patches.

SYNONYMS.—Leukoplakia buccalis; Leucoma; Chronic epithelial stomatitis; Glossodynia exfoliativa; Ichthyosis of the tongue; Psoriasis of the tongue; Keratosis; Tylosis.

Etiology.—The affection is unknown in childhood, rare in women, and generally attacks adult males. The predisposing influences are unknown, although syphilis and gout have been claimed as causes. Tobacco-smokers and glass-blowers sometimes develop it, apparently as the direct result of heat. The presence of broken teeth or the contact of highly-seasoned food, alcohol, or other local irritants appear to be the cause at times. The ingestion of mercury has been claimed to produce it.

Pathology.—The nature of the affection has been considerably discussed. The conclusion has been at last reached that it is neither a psoriasis nor an ichthyosis. The first alteration which takes place is hyperæmia, which is followed by a small-celled infiltration of the superficial layers of the corium, with widening of the blood-vessels and flattening of the papillæ of the Malpighian layer, while the epithelium becomes much thickened and opaque. There is, in fact, a process of hyperkeratosis. This produces patches, at first reddened, but soon grayish-white in color, which scarcely project above the level of the surrounding mucous membrane. Later the patches grow thicker and harder and of a more silvery or yellowish-white color. They are fissured or even warty in appearance, from one and a half to two inches long, sometimes confluent over considerable areas, situated principally on the tongue, lips, cheeks, gums, palate, uvula, pillars of the fauces, and the tonsils.

Symptomatology.—Subjective symptoms may be entirely absent, or there may be unpleasant or painful sensations on mastication or deglutition, and even on talking. This is especially the case when fissures develop.

Diagnosis.—The disease is most apt to be confounded with some syphilitic affection of the mouth. The latter is to be distinguished by the history of the case, the shorter duration of the affection, the presence of ulceration, or other distinctly syphilitic lesions in the mouth or elsewhere, and especially by its yielding to specific treatment.

Prognosis and Course.—The duration of the disease is very prolonged. In fact, the prognosis as regards the removal of the affection is unfavorable. It is, indeed, worse than this, for there is a strong probability of the disease becoming epitheliomatous.

Treatment.—The source of local irritation must be sought for and removed. The exhibition of arsenic has been recommended. Cauterization has been advised, but is apparently capable of increasing the trouble. Mouth-washes of borax, salicylate of sodium, or chlorate of potash may be employed. If the patch assumes a warty condition, the change to epithelioma may be suspected, and surgical interference is to be considered. I have in several instances directed excision on account of the annoyance caused to the patient, even prior to the warty change, and the result has seemed satisfactory.

DISEASES OF THE TONGUE.

The involvement of the tongue in such affections as aphthous, ulcerous, and gangrenous stomatitis has already been alluded to. Certain congenital deformities, as macroglossia and ankyloglossia, and such affections as anthrax of the tongue and new-growths of the organ, belong too strictly to the domain of surgery to discuss them in this connection. There remain, however, certain disorders which deserve independent consideration.

Glossitis.

Definition.—An acute or chronic inflammation of the surface or the parenchyma of the tongue.

Etiology.—Inflammation of the tongue may be conveniently divided into acute and chronic superficial glossitis, and acute and chronic parenchymatous glossitis. The causes of the affection are various. Often a superficial inflammation attends disorders of the mouth, pharynx, or tonsils, or it may depend upon gastric affections, various febrile or other diseases, the development of neoplasms, or the occurrence of trauma arising from broken teeth, corrosive substances, and the like. A unilateral glossitis has been supposed to depend upon some irritation of the chorda tympani. In such cases the inflammation may be attended by the production of vesicles.

A persistent irritation from chronic gastro-intestinal disturbance may produce a superficial glossitis which is chronic from the outset. Chronic alcoholism is a frequent cause of this form. More rarely the chronic variety is consecutive to the acute form of the disease.

In the case of acute parenchymatous inflammation of the organ the same causes may operate, or there may be a powerful etiological element found in the existence of impaired health, exposure to damp or cold, or great fatigue. It may come on during convalescence from acute febrile disorders, or in the course of such affections as rheumatism, scurvy, gout, erysipelas, syphilis, tuberculosis, or poisoning by mercury.

Sometimes persistent local irritation gives rise to circumscribed chronic glossitis, or less often a diffuse chronic inflammation may be the sequel of an acute parenchymatous inflammation of the tongue.

Pathology.—When the inflammation is confined to the surface, the epithelium increases in thickness, and is apt soon to become detached unless it hardens into a crust. After its separation the surface of the tongue is of a bright-red color, with the papillæ unusually prominent. In the chronic form of superficial glossitis there occurs a small-celled infiltration in the mucous membrane which fills up the space between the papillæ and apparently obliterates these. Furrows form here and there which penetrate the thickness of the mucous membrane, dividing the tongue into irregular areas. In some cases shining, smooth oval patches, denuded of papillæ, are scattered over the tongue.

If the body of the tongue be involved, there takes place a rapidly developing congestion of the whole organ, followed by exudation. Resolution generally follows, but the process may go on to suppuration. In the chronic parenchymatous variety there occurs an overgrowth of the connective tissue and a consequent atrophy of the muscular fibres. In the milder superficial cases there is pain on moving the tongue for swallowing or talking, and increased heat and dryness of the organ, but there are no constitutional symptoms.

Symptomatology.—In chronic superficial glossitis the symptoms are often very slight, though occasionally the pain in eating is severe, and the patient is annoyed by a sense of burning irritation. In acute parenchymatous glossitis there occurs an enormous and painful swelling of the whole tongue, which develops within a few hours. Usually the swelling begins at the root, but sometimes the tip or a portion which may have received an injury is first involved. The tongue fills the buccal cavity, and even protrudes beyond the lips and depresses the jaw. Speaking is impossible, swallowing becomes very difficult, and respiration is occasionally dangerously interfered with by the pressure upon the epiglottis and soft palate. The coating of the extruded portion of the tongue is dry, and the organ is hard, immovable, and very painful. The sublingual glands are often swollen. The breath has a fetid odor, there is an increase of saliva, the expression of the face is anxious, sleep is disturbed, and there usually is decided fever. Resolution generally begins in a few days, and is soon complete, although some superficial ulceration is often left. Sometimes the inflammation goes on to the formation of an abscess, this being indicated by an increase in the severity of the symptoms and the development of a softened, circumscribed swelling in some portion of the tongue. Occasionally more or less extensive gangrene results.

In chronic parenchymatous glossitis there is no pain and no constitutional disturbance. The tongue may be somewhat, although not greatly, enlarged throughout, or there may be but a localized enlargement. A certain amount of atrophy may subsequently take place. There is slight pain and some difficulty in deglutition and articulation.

Diagnosis.—This is nearly always easy. Suppurative glossitis, if localized and of slow course, may be taken for a cystic tumor, and circumscribed induration may resemble epithelioma. The history of the case, or, if necessary, aspiration, will solve the question in the first instance, and the discovery of a broken tooth aid the diagnosis in the second.

Prognosis and Course.—The prognosis of acute superficial glossitis is in every way favorable unless the disease pass into the parenchymatous form. That of chronic superficial glossitis is quite the reverse, inasmuch as the lesion usually depends upon some dyspeptic disorder which is apt to prove rebellious to treatment. The prognosis of acute parenchymatous glossitis is usually favorable, the inflammation subsiding in a few days. Death, however, may occur within forty-eight hours. If suppuration takes place, the duration of the affection is more prolonged, but the prognosis is still usually good. The development of gangrene adds greatly to the gravity of the case.

The prognosis of chronic parenchymatous glossitis is good if the cause can be removed.

Treatment.—In acute superficial glossitis attention must be given to the removal of any possible cause. For local treatment may be employed cool, demulcent washes of slippery elm, quince-seed, and the like, If these do not answer, astringent applications may be made. The sucking of small pieces of ice and the use of other means, as advised for the treatment of catarrhal stomatitis, are appropriate. Chlorate of potash is useful when the inflammation depends upon mercurial poisoning.

The treatment of chronic superficial glossitis consists principally in an effort to remove the cause. In addition, it is necessary to keep the tongue very clean, in order that remnants of food do not lie in the furrows and produce irritation. Astringent solutions, such as tincture of myrrh, sometimes give relief.

The treatment of acute parenchymatous glossitis may be started with the administration of a saline purge and confinement to bed. If the tongue be already much swollen, very active treatment is necessary. Free leeching under the jaw should at once be applied, and should this not quickly answer, one or two long incisions should be made into each half of the tongue. Washes and sprays of borax, bicarbonate of sodium, chloride of ammonium, or the like should be employed frequently to keep the tongue moist and to remove inspissated secretion and epithelium. Aconite and, possibly, antimony should be administered internally, with opium if necessary to relieve pain. Tincture of the chloride of iron, given internally in large and frequently repeated doses, has been used in most of my cases, and I have been so fortunate as not yet to have seen suppuration supervene. If it be impossible to introduce food into the mouth, the patient may be nourished through a nasal tube or by enema. An abscess should be opened as soon as discovered.

Localized chronic parenchymatous glossitis is to be treated by the removal of the irritating cause, by the local application of tincture of iodine, and by the internal administration of potassium iodide, with or without small doses of mercury.

GEOGRAPHICAL TONGUE.

Definition.—An affection of the tongue characterized by the development of circinate patches, which creep from one part of the surface to another.

SYNONYMS.—Lichenoid of the tongue; Wandering rash; Pityriasis of the tongue; Eczema of the tongue; Desquamative syphilide of the tongue; Circinate eruption; Ringworm of the tongue; Marginate exfoliative glossitis.

Etiology.—The name selected is apt, in so far as it indicates neither the cause nor the nature of the disease. These are, in fact, unknown. The disease is common in young children, but infants and adults may be affected by it. It has frequently been claimed that syphilis may produce it, but this view cannot be substantiated. Gout has been assigned as the cause, but without much reason. It has been proved not to be of a parasitic nature, as once believed. It is certainly frequently associated with dyspeptic disturbances, and may disappear only with the cessation of the symptoms, to return again with another attack of indigestion. It is so often present in chronic gastric and gastro-intestinal catarrh in children that some authors describe it as a symptom of these affections. On the other hand, it sometimes persists even after the state of the digestion seems normal, and it may be present in apparently healthy individuals. Heredity has been claimed by some to exercise a predisposing influence. The peculiarities of this affection have led me to adopt the view that it is in most instances a tropho-neurosis connected with a morbid state of filaments of the pneumogastric nerves.

Pathology.—The disease begins as one or more round, slightly elevated, flattened whitish spots situated nearly always on the dorsum of the tongue. These spots are produced by a rapid development and heaping up of poorly-formed epithelial cells. Such a patch begins to enlarge, always with circinate, elevated, whitish edges formed of similarly heaped-up cells. Meanwhile, the cells in the central portion are rapidly thrown off, leaving a red, glazed surface which has lost its filiform papillæ, although the fungiform are still present. The edges of the eruption gradually spread, while the central portion becomes re-covered with healthy epithelium. Several patches may run into each other, thus forming waving, sinuous outlines which gradually move from place to place. Thus the tongue may heal in one place, only to be again invaded by the wavy line of the disease advancing from another quarter, or by a new patch which has developed in the re-formed epithelium. In some cases the patches are confined to one side or the other of the tongue, and within the affected area may recur frequently at irregular intervals, or a single patch may persist for many weeks. It has been stated by some that only the epithelial layer is involved in the disease, while others maintain that the primary disturbance is below this. Numerous leucocytes have been discovered in the upper layers of the corium around the vessels.

Symptomatology.—Apart from the condition of the tongue described, and from the manifestations of indigestion so commonly present, there are usually few symptoms to be discovered. Itching of the tongue and salivation have been mentioned as symptoms, but must certainly be very unusual. In patients who are liable to this condition I have very often observed a close connection between exacerbations of gastric catarrh from cold or indiscretion in diet and the recurrence of one or more glazed patches on the tongue, though, as above indicated, the patches may persist after all marked gastric symptoms have subsided. Not rarely I have noted perversion of appetite, especially in the form of morbid cravings, though in no wise differing from what is seen in chronic gastric catarrh unattended with this special lingual condition.

Diagnosis.—The affection can scarcely be confounded with anything else, unless it be certain syphilitic diseases of the tongue.

Prognosis.—The disease is fortunately entirely harmless, for it is very resistant to treatment. Although it frequently disappears quickly, it is exceedingly apt to return. It finally disappears of itself, except in those instances in which it persists into adult life.

Treatment.—Any defect in the general health of the patient should be corrected. Especially is this true of gastro-intestinal disorders. Strict attention should also be paid to every detail of personal hygiene. Apart from this, treatment is scarcely indicated. In obstinate cases, satisfactory results have followed the cautiously prolonged use of small doses of silver nitrate, given in solution on an empty stomach.

NIGRITIES.

Definition.—An affection of the tongue characterized by the development of a black discoloration of the dorsum.

SYNONYMS.—Black tongue; Parasitic glossitis; Glossophytia.

Etiology.—The affection is a rare one, and comparatively little has been written concerning it. The cause is probably some defect of nutrition, but the nature of this is unknown. The disease has been seen in dyspeptics, syphilitics, and hypochondriacs, but it is questionable whether any causative relation exists. In a marked case seen recently, the patient was a highly neurotic young woman, with infantile uterus, neurasthenia, and obstinate gastric catarrh. It may occur at any age.

Pathology.—The nature of the affection is also not well understood. Several writers have claimed that it is due to some parasite. The process seems to consist in a great elongation of the filiform papillæ, with a thickening and hardening of their epithelial layer, which becomes stained a brown or black color. The altered papillæ much resemble hairs. The patch starts in the centre of the dorsum, and its color shades off toward the edges. It gradually increases in size, and is followed by desquamation.

Symptomatology.—The affection produces no subjective symptoms. The black color and the great length of the papillæ are particularly to be noted on inspection.

Prognosis and Course.—The disease may last for months or years. It is generally amenable to treatment, but is liable to recur.

Treatment.—Frequent application of peroxide of hydrogen or a solution of borax is of service. The tongue may also be gently scraped. It has been recommended by Brosin to rub the patch with spiritus saponis kalinus, and to follow this with a 5 per cent. ointment of salycilic acid, or to paint the tongue with a 5 per cent. salycilated collodion. The associated general and gastric condition requires attention.

SYMPTOMATIC AND SECONDARY AFFECTIONS OF THE MOUTH AND TONGUE.

Many of the disorders already described are in a sense secondary, depending, as they so often do, on diseases of other regions. Nevertheless, they are so characteristic that they form entities of themselves, and deserve the separate consideration which has been given them. A number of other conditions can best be grouped under the heading of this section.

The *size and shape of the tongue* vary with both local and general causes. The enlargement in glossitis has already been discussed. In some digestive disorders and in anæmia the tongue is flattened, widened, and indented by the teeth. Macroglossia is often an indication of congenital mental defect, such as idiocy. Diminution in the size of the tongue is a common symptom, especially in severe cases of typhoid and other acute fevers, when it is an unfavorable sign. Unilateral atrophy is generally the result of unilateral paralysis. It has been seen also in locomotor ataxia.

The *movements of the tongue* take place with difficulty in patients exhausted by prolonged febrile diseases. Trembling occurs under these conditions, and is also present in alcoholism, lead-poisoning, and paralysis agitans. Irregular jerking movements are witnessed in chorea. Bilateral and unilateral paralyses of the organ may result from cerebral or bulbar disease.

Anæsthesia of the mucous membrane of the mouth and tongue may exist. If total, the cause is usually cerebral and bulbar. Hemianæsthesia is more common. It depends on hysteria or on some affection of the brain or nerves. Hyperæsthesia is usually the result of local disorder. Loss of taste may be either of local origin or due to cerebral or peripheral nervous diseases.

The *color of the mucous membrane* of the mouth and tongue is often a guide to the primary distant cause. Jaundice shows upon the mucous membrane. Pallor of the mouth is produced by all the causes of anæmia. The influence of lead in producing a blue coloration of the edges of the gums is well known, and a similar green line is said to occur in chronic poisoning by copper. Some years ago, I called attention to the occurrence of a bluish line on the gums as an early, if not actually the first, evidence of argyria. The cyanosis of general widespread venous congestion is seen, of course, in the mouth as well as elsewhere. Besides this, there is a blueness of the tongue and oral cavity present in pertussis. The same condition may be seen in measles. A black or purplish discoloration of the mucous membrane of the mouth in the form of patches is found in Addison's disease and melanosis, and, as already stated, in nigrities.

The *condition of the teeth* is of some importance. Sordes upon them is found in adynamic febrile states. Late or irregular eruption occurs in rickets. Early caries is also a symptom of this disease. Syphilis produces the well-known notching of the permanent upper central incisors described by Hutchinson. The transverse and irregular markings seen upon the teeth of some children are often the result of stomatitis, particularly the mercurial variety. Convulsions and severe illnesses have also been assigned as the cause. These transverse furrows are best seen, according to Hutchinson, on the first permanent molars. Scurvy produces gingivitis, which often advances to an ulcerous process. The gums become swollen and purplish, detached from the teeth, spongy, and bleed very easily. Bleeding from the mouth is sometimes in infants the evidence of hæmophilia.

Eruptions may be found in the mouth in various eruptive fevers. In rubeola there is a slight redness of the fauces and a dotted, dull-reddish, minute, papular eruption upon the soft palate. A very similar rash is sometimes seen in rubella. In scarlatina a bright-red, erythematous blush spreads over the soft palate and often over the whole mouth. It is best marked before the rash appears on the skin ; later it is not so visible. Varicella and variola produce their characteristic eruptions on the pharyngeal and oral mucous membrane. Various other eruptions may occasionally occur in the mouth, such as purpura, pemphigus, urticaria, xanthelasma, lichen, etc. Herpes has also been frequently described as occurring in the mouth. It is a question whether in this situation it is not, at least usually, aphthæ. The occurrence of buccal diphtheria of both primary and secondary form, and the invasion of the oral cavity by erysipelas are familiar facts.

Great attention has been given to the character of the *dorsum of the tongue.* It must be borne in mind that various extraneous substances may color the coating of the tongue, and many healthy individuals have constantly a somewhat furred tongue. In most acute diseases, although the tongue may be unusually red at first, at least at the tip and edges, a coating is quickly acquired. In continued fevers, where the mouth is unusually dry, the coating increases greatly in thickness, and may finally form dense, inspissated crusts with cracks of the mucous membrane. This condition is conspicuous in the typhoid state. Sometimes, instead of this, the coating comes away and leaves the tongue red and glazed. In scarlet fever the coating is rapidly shed and the fungiform papillæ swell and give the tongue a red, granular appearance—the "strawberry tongue." The same condition occasionally develops in other diseases.

The irregularly shaped anterior portion of the tongue, destitute of coating, as often seen early in typhoid fever, has been called the "typhoid triangle." I have often noted the same appearance in relapsing fever. A yellowish tinge to the tongue has been considered to indicate hepatic disturbance. It is doubtful whether it has any such significance. In advanced diabetes the tongue is often glazed and shiny and of the color of raw beef. The "irritable tongue," as seen in tubercular peritonitis, tubercular ulceration of the intestines, and sometimes in dyspepsia and in drunkards, is bright red with prominent papillæ. It is similar to the strawberry tongue of scarlatina.

Fissures and ulcers of the mouth and tongue may be present as secondary symptoms. Syphilis is one of the most frequent causes of these lesions. A common condition is the presence of fissures on the lips, especially the upper one, or at the angles of the mouth. Mucous placques and ulcerations are found on various portions of the mucous membrane of the mouth, including the tongue. Fissures of the tongue may of course be syphilitic. It must not be forgotten, however, that they may be merely a symptom of chronic superficial glossitis depending upon chronic dyspepsia. Chancres and gummata may also occur in the mouth.

Tuberculosis also sometimes produces ulceration of the palate or tongue, which may rarely be primary. It is located at the side or tip of the tongue, is irregular in shape, and gradually extends in breadth and depth, without any disposition to heal.

A frequent form of ulceration is that seen in pertussis. It is situated at the frænum, and is found in a large proportion of cases. Gonorrhœal inflammation of the mouth has been occasionally reported, the cases resulting from buccal coitus.

DISEASES OF THE SALIVARY GLANDS.

BY WILLIAM PEPPER.

PAROTITIS.

THE inflammations of the parotid gland may be of two forms: (1) specific parotitis, or mumps, which is described elsewhere; and (2) symptomatic or metastatic parotitis.

SYMPTOMATIC PAROTITIS.—This is usually a rapid suppurative process, the result of some septic infection. It occurs in the course of pneumonia, pyæmia, phthisis, or any severe disease, but especially as a complication of typhoid and typhus fevers. It may be either produced by an infection through the blood, or more often by the ascent through the duct of toxic substances the result of decomposing secretions in the mouth. Sometimes a secondary parotitis occurs, as Paget has shown, in connection with abdominal or pelvic injuries or diseases. I have seen a very severe case with gastric ulcer. The swelling resembles that of mumps, but is generally much greater, and generally also ends in suppuration. The affection is to be treated at the outset by applications of ice, leeches, iodine, mercurial ointment, and similar remedies in the hope of obtaining resolution. If it becomes evident that suppuration must take place, poultices may relieve the pain and hasten the process. Early incision is then required. If a small but deep incision be made, and a drainage tube introduced, with careful antiseptic precautions and with warm fomentations continued to promote suppuration, evacuation may be secured with a minimum of disfigurement.

PTYALISM.

Definition.—A great increase in the secretion of the salivary glands.

SYNONYMS.—Salivation; Sialorrhœa.

Etiology.—Salivation depends upon a great variety of causes. It may be due to a reflex irritation of the salivary nerves, and it is in this way that the symptom is produced in the various forms of inflammation of the mouth already described. The beginning of dentition is accompanied by hypersecretion of saliva, but it is a question whether this is due to the establishment of the salivary function or to a reflex irritation from the advancing teeth. The ingestion of certain drugs, as gold, silver, iodine, copper, arsenic, lead, and particularly mercury, produce it, probably through a reflex irritation by the stomatitis which is set up by them, although it is possibly also through a direct action upon the gland. Irritating substances increase the secretion of saliva by reflex action. So also do diseases of distant organs, such as those of the gastro-intestinal tract. Affections of the liver, spleen, and pancreas have likewise been assigned as causes, and those of the genital organs not infrequently produce it.

Ptyalism may also be the result of a central irritation. Thus, it has been seen occasionally in tumors and diseases of the pons and medulla. Psychic action, as a conception of certain tastes, or psychic disorders, as hysteria or insanity, may be attended by it. The ingestion of certain drugs, especially jaborandi, produces salivation, possibly by an action through the nervous system.

Certain cases of ptyalism in children were reported by Bohn in which the secretion was excessive when the patients were about during the day, but was diminished when they lay down. He believed the condition due to some neurosis.

Symptomatology and Diagnosis.—The chief symptom is of course the constant, excessive secretion of saliva. This seems to be constantly running into the mouth in unmanageable quantities, so that it interferes with talking, necessitates constant swallowing, and often is so great in amount that it runs over the lips. The exact diagnosis of the affection is to be made by the collection and measurement of the secretion in order to prove that there is an actual increase of it. However, for all practical purposes there is no difficulty in diagnosis, except in those cases where certain paralyses interfere with the swallowing of saliva or its retention in the mouth.

Prognosis.—This depends entirely upon the cause, since with its removal salivation ceases.

Treatment.—Effort must be made to discover and remove the cause. The only symptomatic treatment is the employment of atropine.

XEROSTOMIA.

Xerostomia (Aptyalism; Dry mouth), is a suppression of the secretion of the mouth. Its cause is unknown, although it appears in its graver forms to depend upon some nervous influence, such as fright, hysteria, and hypochondriasis, or upon an affection of some unknown centre controlling the salivary and buccal glands. It is also present to some degree in diabetes mellitus, mouth-breathing, febrile states, and sometimes in old age. The symptoms produced by it are dryness and redness of the mucous membrane of the mouth and tongue, with consequent great difficulty in mastication, deglutition, and speech. The tongue may look like raw beef, and be crevassed like alligator skin. Galvanism is sometimes useful for its cure, as is pilocarpine. Frequent moistening of the mouth with water or oily substances gives relief.

ANGINA LUDOVICI.

Ludwig's angina (Cellulitis of the neck; Phlegmonous inflammation of the floor of the mouth) is probably in most cases at first an inflammation of the submaxillary gland. It may arise either primarily or from trauma, or may develop as a secondary inflammation in the course of a severe disease. It starts in the neighborhood of the submaxillary gland on one side, and rapidly spreads over the front of the neck and the floor of the mouth, producing great impediment to mastication, swallowing, and speaking. Great dyspnœa may develop. Unless treatment be prompt and energetic severe general infection is apt to take place. The process may result in the formation of an abscess which may break into the mouth or externally on the neck, or a widespread gangrenous destruction of the involved tissues may occur.

The prognosis of the case is always a doubtful one. At the beginning of the disease efforts should be made to obtain resolution by the application of leeches or by ice, but as soon as pus begins to form thorough surgical interference becomes necessary.

DISEASES OF THE PHARYNX AND TONSILS.

By WILLIAM PEPPER.

DISEASES OF THE PHARYNX.

Acute Catarrhal Pharyngitis.

Definition.—An acute superficial inflammation of more or less of the mucous membrane of the pharynx, tonsils, and palate.

Synonyms.—Sore throat; Angina simplex; Catarrhal angina.

Etiology.—The disease may occur at any age, though perhaps it is most frequent in young persons. Certain individuals, though otherwise healthy, exhibit a remarkable predisposition to it, and this tendency may show itself in several members of one family. A debilitated constitution, anæmia, imperfect hygiene, and the like very commonly predispose to it. Among the exciting causes exposure to cold and wet is by far the most frequent. Rheumatism is also so often the direct cause that "rheumatic angina" has been described. Gout, too, may show itself in the throat. Some of the exanthemata, particularly scarlatina and rubella, are nearly always accompanied by it. Facial erysipelas is likewise often attended by it, and the pharyngitis may then be simply an extension of the inflammation of the face. It is seen in variola, rubella, typhoid and relapsing fevers, and occasionally in pneumonia. Malaria sometimes acts as a cause, and syphilis as a very common one.

Pharyngitis may occur epidemically, and may then be due to certain conditions that are not understood or to some equally unknown infection. It may also arise from trauma, as from swallowing pieces of bone or other hard bodies, or from the action of corrosive liquids, the ingestion of food that is too hot or too cold, or the inhalation of steam or irritant vapors or gases. Certain drugs may cause it, such as copper, mercury, bromine, iodine, antimony, silver, gold, lead, belladonna, or stramonium. Finally, the process may attend similar inflammation of the mouth, larynx, and nose.

Pathology.—The process is at first simply a hyperæmia, with dilatation of the veins, and may not go beyond this. In the severer cases there occurs an inflammatory exudation, with swelling of the tissues. The mucous glands are swollen and secrete abundantly. The uvula is often swollen and adherent to one or the other of the sides of the palatine arch. The process may be diffuse or only certain portions may be involved. The affected mucous membrane appears abnormally red, glistening, and either dry or covered with a viscid mucus.

Symptomatology.—Except in the mildest cases the disease usually begins with fever, the grade of which depends upon the severity of the attack. There may even be a chill at the onset, but this is more frequent if the tonsils are much involved. There is a feeling of soreness in the throat, made worse by the effort to swallow, and often very severe, and there is also a constant sensation of tickling, which causes a desire to cough or hawk. Talking is frequently painful, and the voice harsh from the secondary extension to the larynx. Occasionally the neck is stiff and the lymphatic glands tender and enlarged. The symptoms are usually worse on one side, but in a few days after convalescence is established the other side is very liable to be attacked. The hearing is often a little dull, there is pain in the ears, and the voice is somewhat nasal. In the rheumatic form the pain in swallowing often seems out of proportion to the condition of the mucous membrane seen on inspection, and the disease is liable to pass off suddenly, and to be followed by rheumatic manifestations elsewhere in the body.

Diagnosis.—This is a matter of no difficulty. Inspection determines whether we have to do with a pharyngitis or a tonsillitis. A fact very important to bear in mind is that pharyngitis is often the first, and sometimes the only, local symptom of scarlatina.

Prognosis and Course.—The prognosis of idiopathic cases is entirely favorable. The mildest form recovers in twenty-four hours, and at the longest the disease lasts from a few days to a week. There often remains a decided disposition to recurrence.

Treatment.—Prophylactic treatment consists of avoidance of close rooms and too sedentary an occupation. The employment of cold bathing, especially of the throat and chest, is often extremely useful. During an attack the patient should be confined to the house, and, if there be much fever, to bed. A saline purge or divided doses of calomel are indicated at the outset. Pieces of ice may be sucked with relief. If there be much fever, aconite may be administered with advantage. Excellent results are obtained by the administration of a combination of the tincture of the chloride of iron and chlorate of potash. If rheumatism be suspected as the cause, full doses of benzoate of sodium or salicylate of sodium will often give prompt relief. Guaiacum has been highly recommended for rheumatic and other forms. Astringent solutions and anodyne lozenges are of great benefit. The throat may be painted with solutions of nitrate of silver or tannic acid or with the tincture of the chloride of iron. At the same time any constitutional cause must be appropriately treated.

CHRONIC PHARYNGITIS.

Definition.—A chronic inflammation of the mucous membrane of the pharyngeal structures, diffuse or circumscribed, and affecting different elements in the pharyngeal tissue.

SYNONYMS.—Chronic catarrhal pharyngitis; Chronic follicular pharyngitis; Ulcerative sore throat; Granular pharyngitis; Chronic angina; Clergyman's sore throat; Chronic catarrh of the throat; Pharyngitis sicca.

As indicated by the number of synonyms, several somewhat diverse varieties of pharyngitis are included. The classification of these forms varies with different writers. Probably one of the simplest is that which divides them according to the relative degree of involvement of the glandular structures. We may then have—(1) *Chronic catarrhal pharyngitis* (chronic catarrh of the throat, "relaxed throat"), in which the inflammation is diffuse, superficial, and with but slight involvement of the follicles. The "relaxed sore throat" is perhaps one of the mildest forms of this. There exist in it a congestion and swelling of the mucous membrane of the fauces, with a certain lack of tone about the tissues. (2) *Chronic follicular pharyngitis* (granular pharyngitis, "clergyman's sore throat"), in which the inflammation is largely confined to the follicles. Each of these two conditions may also exhibit (3) an atrophic form, the *pharyngitis sicca*. Whether the atrophic form of chronic follicular pharyngitis is always a later stage of the hypertrophic form, or whether it is sometimes an entirely distinct process—the exudative form of chronic follicular pharyngitis, described by Mackenzie—is not yet clearly determined.

Etiology.—Chronic pharyngitis is less common in children than at a later age. Men are more often affected than women, probably on account of a greater degree of exposure to the exciting causes. Improper hygiene, the existence of rheumatism, gastric disorders, gout, phthisis, and other chronic diseases, and particularly a condition of general anæmia and debility, strongly predispose to it. Heredity exerts some influence. The disease is also often associated with chronic inflammation of other mucous membranes.

Among the exciting causes one of the commonest is the occurrence of repeated attacks of acute catarrhal pharyngitis. Another very common cause, particularly of the follicular variety, is excessive or uneducated use of the voice in those in whom some debility of constitution already predisposes. A too free use of alcohol and excessive tobacco-smoking are powerful causes of the catarrhal form with relaxed throat. Among other causes of chronic pharyngitis may be mentioned the inhalation of dust and of irritating gases, and the presence of venous congestion resulting from chronic pulmonary and cardiac diseases. It has also been said to arise by a reflex irritation in cases of uterine disorders. Both disease of Luschka's tonsil and intranasal affections have been claimed to be the exciting cause of general chronic pharyngeal catarrh.

Pathology.—In *chronic catarrhal pharyngitis* there is present a hyperæmia of the mucous membrane, particularly of the posterior pharyngeal wall, the posterior pillars of the fauces, and the soft palate. With this is serous infiltration and some increase of tissue-elements, and later there develops a permanent varicose condition of the smaller vessels, which often maps the pharynx into small irregular areas. The diseased mucous membrane appears bright red and unevenly thickened. The glands are enlarged, but not to any considerable extent. There is hypersecretion from the diseased area, which consequently often appears peculiarly shining from the coating of viscid mucus. The palate and fauces are relaxed and the uvula elongated.

In *chronic follicular pharyngitis* the process affects principally the mucous follicles, which become much hypertrophied and distended. They appear as small, isolated elevations from the size of millet-seeds up to that of peas, which are dotted principally over the posterior pharyngeal wall. They are either of a yellowish-white or of a deep-red color, and are surrounded by a network of dilated venules. Tenacious discolored mucus coats the membrane. As the process advances the elevations become more numerous, and coalesce to some extent. The posterior pharyngeal wall often assumes a reticulated appearance, and ridges made by the fusion of the elevations are seen in places, particularly close behind the posterior faucial arch. The disorder gradually spreads, and finally involves much of the mucous membrane of the pharynx and mouth. As in the catarrhal variety, the uvula is apt to become elongated and the palate relaxed.

In either form of chronic pharyngitis the mucous membrane has its function interfered with, and finally undergoes atrophy. The secretion is then scanty, and forms crusts upon the mucous membrane, which appears much thinner than normal, glazed, and dry. The variety described as exudative exhibits follicles filled with milk-like or cheesy secretion, which often exudes and coats the pharynx in small, white, irregular patches. A tendency to atrophy is observed from the beginning.

Chronic pharyngitis may produce ulcerations, but this is rare.

Symptomatology.—The principal symptoms of chronic pharyngitis are cough, huskiness and loss of power of the voice, stiffness, dryness, and uncomfortable sensations in the throat, a disposition to clear the throat, and more or less pain in swallowing. These symptoms vary in different cases according to the condition of the throat. They may last through the whole day or be present only in the morning or the evening. In the catarrhal variety, if great relaxation of the palate be also present, the tickling cough is sometimes distressing, and may induce vomiting. The amount of hawking depends largely on the quantity and viscidity of the secretions. It is usually worse in cases complicated by post-nasal catarrh, owing to the descent of the secretion into the oral pharynx. Sometimes the first sign of the follicular variety is the affection of the voice, and sometimes, too, the disease exists a long time without any symptoms referred to the throat, and is accidentally discovered by the physician.

Diagnosis.—The diagnosis presents no difficulty. The persistent cough with expectoration, which is sometimes bloodstained, occasionally arouses the suspicion of phthisis, but the examination of the throat explains the source of the symptoms.

Prognosis.—The prognosis of the catarrhal form is favorable if the cause of its continuance can be removed. That of chronic follicular pharyngitis is much less so. If the disease be not too far advanced, treatment can remove most of the disagreeable symptoms. The ability fully to restore the voice, however, is very questionable. The atrophic form is still more resistant to treatment.

Treatment.—In any form of chronic pharyngitis it is primarily important to institute treatment directed to the removal of the cause, for unless this be done but little can be expected from local measures. Rigid attention is demanded to personal hygiene in order to improve the tone of the system and to avoid recurring attacks of acute catarrh. The patient must be instructed to avoid mouth-breathing; nasal obstructions must be removed; and systematic diaphragmatic breathing and proper elocution must be insisted upon. For the catarrhal variety of the disease mildly astringent lozenges or direct applications are useful. Sprays of hamamelis, chloride of ammonium, boric acid, tannic acid, sulphate of zinc (5 grains to the ounce), and the like are useful. In more obstinate cases stronger astringents must be applied by a brush; among these being the tincture of the chloride of iron and strong solutions of tannin in glycerin. If the uvula remain persistently elongated, it may be necessary to cut off a portion of it.

In the follicular variety the same kind of local treatment may be employed in the mildest cases, but, as a rule, more energetic procedures are necessary. The diseased follicles should be carefully destroyed by such caustics as chromic acid, London paste, nitrate of silver, or the galvano-cautery. A single application is usually sufficient for each follicle. Very few follicles, possibly only one, should be touched at one sitting, and the sittings may be repeated every few days. In the intervals applications can be made of solutions of chloride of zinc, chloride of iron, or iodine. If the throat become much irritated, sedative applications, such as alkaline sprays or the inhalation of benzoin are indicated.

In the atrophic form measures must be taken to keep the throat moist. Chloride of ammonium, given in lozenges, sprays, or internally, promotes secretion, as does jaborandi. Frequent spraying with hot water, glycerin and water, or some weak alkaline liquid, as Dobell's solution, will also give relief.

ULCEROUS PHARYNGITIS.

This is a low grade of inflammation of the pharynx, probably of a septic nature, characterized by the development of one or more white superficial ulcers on the pharynx, tonsils, or palate. The ulcers are of varying size, round or oval, and tend to become confluent. The disease is apt to occur in those who are debilitated from some cause, as through lack of rest, inadequate exercise, or lack of fresh air. It is also seen in those exposed to septic influences, as the air of hospital wards or of the dissecting-room. Sometimes slight ulceration of the tonsils remains as the result of a follicular tonsillitis.

The chief local symptoms consist of severe pain in the throat, made worse on swallowing. The tongue is coated and feels stiff, the breath is heavy. The constitutional symptoms are marked; and consist in debility, loss of appetite, weak pulse, fever, headache. The ulcers heal as the general health improves.

Treatment is chiefly constitutional, the tonics, and especially quinine and iron, being indicated together with removal from the unhealthy surroundings. Locally astringent and antiseptic applications, such as peroxide of hydrogen properly diluted or a saturated solution of iodoform in ether, may be used.

Ice may be given to relieve pain if necessary. Warm inhalations, as of vapors of benzoin, are sometimes very soothing in their effects.

MEMBRANOUS PHARYNGITIS.

Membranous pharyngitis (Herpetic pharyngitis; Herpetic tonsillitis; Aphthous pharyngitis) appears to be the identical in nature with aphthæ of the mouth, the locality only being different, and therefore needs no extended description here. It is an unusual affection, particularly liable to develop in women and children and delicate individuals. The exciting cause of the disease is not certainly known. Exposure to cold, cold and damp climates, uterine diseases, nervous influences, local irritation, and the prevalence of diphtheria have each been assigned as the cause.

Vesicles appear, a few in number or in successive crops, upon the mucous membrane of the pharynx, soft palate, pillars of the fauces, tonsils, and often of the tongue and cheek. This vesicular stage is rarely seen. The vesicles soon rupture, and form small, circular, superficial ulcers, which resemble the aphthous ulcer described. In the severer cases these may coalesce and form areas somewhat resembling diphtheria.

Some malaise and fever usher in the development of the pharyngeal condition. There is next soreness of the throat, which is often very severe, and which is made much worse by swallowing; salivation is frequent, and there is loss of appetite. After four or five days the ulcers heal and recovery is complete in about two weeks.

The disease when severe may be confounded with diphtheria. In such cases it can only be distinguished by the discovery of some fresh, small discrete lesions which have not taken on the diphtheritic appearance, or of others which, although already covered by false membrane, yet by the translucency of this and by their circular shape indicate their original source.

The treatment consists in febrifuges containing aconite if the temperature demand it. A saline laxative may well be given. Sedative antiseptic sprays and washes are of value. Among them may be mentioned solutions of permanganate of potash, alum, borax, myrrh, and chlorate of potash. In the severer forms tonics and stimulants are indicated.

PHLEGMONOUS PHARYNGITIS.

In the most common form of this disease the process is located in the soft palate, usually to one side, and in the uvula, and even extends into the hard palate. The nature of the inflammation and the course of the symptoms and of the case in general are practically identical with those of quinsy, with which it is usually associated. The affection is, in fact, a peritonsillar abscess, and is best described in connection with parenchymatous tonsillitis.

A very infrequent form, unassociated with tonsillar inflammation, is the deep-seated suppurative pharyngitis which is prone to become diffuse, the process spreading downward beside the œsophagus or anteriorly about the trachea into the neck. It may be the result of some injury, as from a foreign body in the pharynx or from the swallowing of scalding or corrosive liquids or the inhaling of steam; or it may follow some severe infection. The grave cases of erysipelatous pharyngitis may be best included here.

The process is an intense one. The constitutional symptoms, which are severe, are often ushered in by a chill. The fever is high, the throat very painful, swallowing difficult, and dyspnœa often present from involvement of the larynx. The disease is prone to become gangrenous if the patient live long enough, and in such case more or less of the mucous membrane sloughs.

The prognosis is grave. If recovery takes place, cicatrization with stricture may remain.

In the line of treatment every effort must be made at the outset to abort inflammation. Aconite should be given in full and repeated doses, or, if the affection be still more threatening, leeching may be employed. Ice should be held in the mouth and cold compresses applied to the neck. Opium will relieve pain. Food may be administered by enemata if swallowing be too painful. The general treatment should be strongly tonic.

Gangrenous Pharyngitis.

This process may attach itself to any preceding form of pharyngitis, or it may, and most commonly does, develop independently of any other. It may follow any of the eruptive diseases, especially erysipelas and scarlatina, or it may occur idiopathically. The pharynx, palate, and tonsils become livid and swollen, and dark-gray or blackish ulcers with excavated edges form on them and rapidly extend.

The symptoms quickly become those of extreme depression of strength. There is a low grade of fever, cold extremities, rapid, feeble pulse, often diarrhœa, little pain, a gangrenous odor of the breath, and possibly hæmorrhages from various parts of the body, including the diseased pharynx. Death usually results. Sometimes erosion of the carotid takes place. Recovery is prone to be accompanied by great deformity.

The treatment consists in thorough cauterization of the affected parts whenever this is feasible. Antiseptic and anodyne lotions and sprays are advisable as palliative measures. Internally tonic remedies and stimulants are indicated. The difficulty in swallowing interferes greatly with medication and with sustentation. Enemata, both nutritious and medicated, may be required; and cardiac stimulants may be used hypodermically.

Mycosis Pharyngis.

In this very unusual affection—also called mycotic pharyngitis—there develops, principally on the crypts of the tonsils and on the base of the tongue, a peculiar yellowish or white deposit, more or less indurated and sometimes very hard, and tough, and tenacious, and which is reproduced rapidly after removal. The masses are formed of filaments and spores, which are found to be those of *Leptothrix buccalis.* The organism penetrates to the deeper parts of the mucous layer, though it is sometimes only superficially situated. There is no local evidence of inflammation found, nor are the symptoms witnessed usually other than slight tickling sensations in the throat. Sometimes even this is absent, while in other cases there may be actual difficulty in swallowing and pain.

The diagnosis can be made by the microscope. Apart from this, the disease may readily be distinguished from follicular tonsillitis and diphtheria by its chronic course and by the absence of constitutional symptoms. The affection is a harmless one, but the prognosis so far as cure is concerned is very dubious. The disorder is very persistent and little amenable to treatment. The fungus should be scraped away with a curette and its former seat cauterized. Antiseptic solutions should also be frequently applied to the parts.

RETROPHARYNGEAL ABSCESS.

Definition.—A deep-seated suppurative inflammation of the submucous tissue of the pharynx.

SYNONYM.—Retropharyngeal lymphadenitis.

Etiology.—The disease is generally one of childhood, although it may even attacks infants, and sometimes is seen in adults. Many cases seem to be idiopathic, although the scrofulous diathesis or some constitutional debilitating disease may have pre-existed. The general hygienic conditions and the existence of diseased states of the naso-pharynx are also predisposing factors. The disease may occur after zymotic affections, especially scarlatina, measles, and erysipelas. Trauma may also induce it, and it may be secondary to caries of the cervical spine.

Pathology.—In the majority of cases the disease arises as an inflammation of the retropharyngeal lymphatic glands. The affected glands pass through the usual stages, and finally form an abscess, which is situated in the submucous connective tissue and lies in front of the periosteum and the vertebræ, and generally more to one side. When the disease arises from a spondylitis the anterior wall of the vertebræ is destroyed, and the inflammation spreads by contiguity from the osseous structures. The pus tends to burrow in any direction, but especially downward.

Symptomatology.—The onset of retropharyngeal abscess is generally insidious, and the first symptoms do not indicate its presence. Often there is no positive indication of the affection until the tumor has attained sufficient size to interfere with swallowing or with breathing. In young children there is an increasing disinclination to swallow nourishment offered, while older children complain of pain in the pharynx and stiffness of the neck, with pain on moving the head. The respiration often is noisy, rattling, irregular, and through the open mouth, and the voice is altered in character. The patients are pale and restless, and convulsions are very apt to develop in young subjects. There is swelling and tenderness of the sides and front of the neck. Palpation of the pharynx with the finger reveals a tumor, more or less soft according to the stage which it has reached. The symptoms depend somewhat upon the exact position of the abscess. If it be high in the pharynx, respiration is not so greatly interfered with and the voice is nasal. If it be lower, opposite the larynx, there occur attacks of extreme dyspnœa, with cough and stertorous respiration. Swallowing also is greatly interfered with, and the voice is hoarse or even absent. If the abscess be situated laterally, the head is drawn toward the healthy side. Sometimes the head is thrown backward. It often happens that the abscess is situated so low down as not to be visible even when the tongue is depressed strongly, but, if the forefinger be pushed as far as possible down the œsophagus, the bulging of the posterior or lateral wall at the level of the abscess may be detected in many instances. If the process be a rapid one, there may be considerable fever, although the temperature in this disease is usually but little elevated. In cases running a slower course fever is always but slight unless some complication exist. Diarrhœa and vomiting sometimes attend, while in other cases the digestion and appetite are good, and only the pain on swallowing prevents the ingestion of food.

Diagnosis.—The diagnostic symptoms of retropharyngeal abscess are interference with deglutition, dyspnœa with noisy mouth-breathing, altered, nasal voice, pain and swelling in the pharynx, swelling, pain, and stiffness in the neck, and possibly the detection of a tumor by digital exploration. Croup, which often greatly resembles it, is to be distinguished by the absent voice and the absence of dysphagia and of swelling of the pharynx. Œdema of the glottis and foreign bodies in the larynx are likewise free from the last two symptoms. The latter affection generally produces greater interference with the voice.

Prognosis and Course.—The course of the disease depends upon the cause. Idiopathic abscess may form with great rapidity, and terminate in three to four days. The symptoms develop quickly, and are often very threatening. Cases following scarlet fever also go on speedily to suppuration; but, as a rule, secondary cases run a longer course, and those dependent upon cervical caries often last for weeks or even months. In these more chronic cases the development of the symptoms is proportionately slower, and the general health of the patient suffers to a much greater degree.

The prognosis is always dubious, and depends upon various circumstances. The majority of the idiopathic cases recover, the discharge of the abscess being followed by immediate relief of all the symptoms. Sometimes death has occurred from suffocation by the pus. The prognosis is unfavorable where the abscess has lasted for some time and interfered much with the general health of the patient, or where the child is already debilitated by some chronic or acute disease. There are always, too, the dangers of the pus burrowing in different directions, especially toward the larynx, where it may produce fatal pressure. Septic absorption is also a cause of danger, as is also the possibility of fatal hæmorrhage. Other things being equal, the more prompt and thorough the treatment the better does the prognosis become.

Treatment.—The constant application of ice externally and internally may be employed if suppuration has not already taken place, but prompt evacuation is indicated as soon as pus has formed. The incision with a guarded bistoury should be made as near the median line as possible, and the head at once bent far forward to prevent the pus passing into the larynx. When deglutition is almost or quite impossible nutritious enemata should be administered. Opium is often required to allay pain and to secure rest. The after-treatment consists in the repeated cleansing of the mouth and of the abscess-cavity, and in the administration of tonic remedies.

DISEASES OF THE TONSILS.

ACUTE SUPERFICIAL TONSILLITIS.

THIS disease—called also acute catarrhal tonsillitis—consists of an acute inflammation of the mucous membrane of the tonsils. It is merely a localization on these bodies of the process described under Acute Catarrhal Pharyngitis. The latter disorder is, in fact, more often localized on the tonsil, or at least better developed there, than on any other of the pharyngeal structures.

In addition to the cause already described, may be mentioned the etiological influence of chronically enlarged tonsils, since these have a great tendency to take on acute superficial inflammation. In a similar way the presence of cheesy or calcareous deposits in the tonsils is productive of tonsillar inflammation. The frequency of the action of the rheumatic poison also deserves special mention.

The disease differs from other forms of tonsillar inflammation in that only the mucous membrane covering the gland is involved. The organ, it is true, is often distinctly swollen, partly as a result of the thickening of the mucous membrane, and partly dependent upon a slight attendant parenchymatous inflammation. It is never, however, enlarged to anything like the degree witnessed in quinsy. The lymphatic glands in the neck are little if at all swollen.

The symptoms are those already described. The preponderating involvement of the tonsils increases the tendency to chill and high fever. The thin grayish coating which sometimes forms consists of epithelium, pus-cells, and mucus, and is easily brushed off. The disease lasts rarely longer than five to eight days, and generally a shorter time.

The treatment is that for acute catarrhal pharyngitis.

ACUTE LACUNAR TONSILLITIS.

Definition.—An inflammation of the tonsils involving the mucous membrane lining the crypts as well as that of the surface.

SYNONYMS.—Acute follicular tonsillitis; Angina lacunaris.

Etiology.—The disease is commonest from the fifteenth to the thirtieth year of age, is not infrequent between the fifth and fifteenth years, and is much less common in young children under five years of age.

The causes are the same as those of catarrhal pharyngitis and laryngitis.

Especially to be mentioned is the influence of some form of sepsis, which often appears to produce the disorder. This is seen, for example, in lacunar tonsillitis following exposure to defective drainage. Lacunar tonsillitis may also be the result of the infection of scarlet fever. Cases, too, appear to be very frequent during epidemics of diphtheria. It is very probable, however, that many of these latter are not lacunar tonsillitis in the ordinary sense of the term, but are instances of the localization of the diphtheritic process upon the tonsillar crypts.

Pathology.—One or both tonsils are decidedly swollen, and their surface reddened and dotted with a number of yellowish-white spots, which correspond to the openings of the lacunæ. These white spots may project beyond the surface. Sometimes the secretion of closely adjacent lacunæ becomes confluent, and appears to form a membranous mass which may simulate diphtheria.

The secretion from the crypts can be easily scraped away, and leaves no ulceration beneath it. It is composed of epithelial and pus cells, bacteria, and detritus, often with some fat-crystals and cholesterin. The swelling of the tonsil is due to some degree of parenchymatous change which is commonly present, and which consists of a serous and cellular infiltration. Ordinarily the secretion is discharged in a few days, and the tonsil returns to its normal condition. Not rarely the contents of some of the lacunæ are retained, and desiccate and even become calcareous. Occasionally the inflammation is severe enough to produce superficial ulceration. With lacunar tonsillitis is always associated some degree of inflammation of the pillars of the fauces and the soft palate. There is frequently enlargement of the lymphatic glands of the neck.

Symptomatology.—The constitutional symptoms of follicular tonsillitis are often very severe. There is distinct chill, high fever with its attendant symptoms, and sometimes a degree of depression of strength which is very striking. The pulse is often extremely rapid. In severe cases occurring in sensitive children mild delirium may be present. There is usually very distinct pain on swallowing and the glands at the angles of the lower jaw are decidedly swollen and tender. The tongue is coated and the appetite is greatly impaired. The urine is strongly febrile, but only in unusually severe cases is a trace of albumin present. In many respects the symptoms are the same as those of acute catarrhal pharyngitis.

Diagnosis.—In typical cases the diagnosis is made without difficulty by inspection of the throat, but in some instances the disease may be mistaken for diphtheria. The deposit, however, in lacunar tonsillitis is not a membrane, is more elevated, more easily removed and does not come off in strips, and the surface below it is not eroded and bleeding. After removal it does not re-form as rapidly as in the case of diphtheria, and, more especially, in the latter affection the membrane is seldom limited to the tonsil. Often, however, it is difficult or impossible to make an early diagnosis. The danger of confusion has been needlessly increased by the fact that this affection has often been spoken of as diphtheritic sore throat. Occasionally the suddenness of the onset, the abrupt rise of temperature, the rapid pulse, the restlessness, the swelling of the tonsils and glands at the angles of the jaw, the pain in swallowing, suggest strongly the possibility of scarlatina in one who has never passed through an attack of the latter disease. It must be remembered that acute lacunar tonsillitis does occur in a certain number of cases of scarlatina. The absence of marked nervous symptoms and the failure in the appearance of any eruption soon clear up the doubt.

Prognosis and Course.—The course of lacunar tonsillitis is always favorable. The duration is three to four days. A tendency to recurrence remains, and one or more attacks may be followed by chronic tonsillar hypertrophy. In some subjects there is a strong tendency to recurrent attacks, and I have had occasion to watch several children in whom from the age of three years there have been, in spite of strict hygienic care, frequent typical attacks, growing less frequent and finally ceasing with the full development of adolescence.

Treatment.—As a prophylactic measure, in view of the occasional difficulty in diagnosis, patients with lacunar tonsillitis should be separated from others. If seen early they should be given a brisk saline cathartic, be confined strictly to bed, and ordered a fever mixture containing aconite. Mild sedatives are required to allay pain and restlessness. Full doses of salicylate of sodium are indicated if a rheumatic cause be suspected. Astringent lozenges or astringent gargles may be employed, and the local use of guaiac is also often of benefit. The direct application of astringent solutions is frequently of service, although these should generally be weak.

Phlegmonous Tonsillitis.

Definition.—An acute inflammation of the parenchyma of the tonsil, frequently going on to suppuration.

Synonyms.—Quinsy; Tonsillar abscess; Suppurative tonsillitis; Parenchymatous tonsillitis; Cynanche tonsillaris.

Etiology.—The causes are similar to those of other forms of tonsillitis. The affection rarely occurs before the age of ten to fifteen years, and not often after forty years. A remarkable individual predisposition sometimes exists to such an extent that while some persons never suffer from the disease, no matter to what exciting causes they may be exposed, others develop it repeatedly. It has seemed to me that in such cases a decided gouty diathesis is apt to coexist. The presence of chronic enlargement sometimes acts as a predisposing cause.

The most frequent exciting cause is exposure to cold. An acute catarrhal tonsillitis already present may advance to a parenchymatous inflammation. Blocking up of follicles by inspissated secretion may likewise set the inflammation in action.

Pathology.—Although there is inflammation of the surface of the tonsil, and often of the lacunæ also, the principal seat is in the glandular tissue. The tonsil becomes greatly swollen by infiltration with inflammatory products. Resolution may next occur, or, if the process be sufficiently severe, one large or several smaller abscesses may form. The palatine folds, the soft palate, and the cellular tissue about the tonsil are involved in the inflammatory process, and an abscess may occasionally form in these regions (peritonsillar abscess, phlegmonous pharyngitis of one form) instead of in the tonsil itself. Œdema of the epiglottis and of the upper part of the larynx and inflammation of the base of the tongue are not infrequent. At the height of the disease the tonsil may become so large that it projects beyond the middle line. The mucous membrane covering it is of a bright-red color, the anterior palatine fold is pushed forward and is stretched over it; and the uvula is often œdematous, red, and adherent to one or the other side. The salivary glands and the lymphatic glands of the neck are often swollen and tender.

Symptomatology.—The disease frequently begins with a chill, which is rapidly followed by high fever. Pain in the throat, especially on swallowing, develops at the same time. There are headache, rapid pulse, malaise, insomnia, and loss of appetite. The pain in the throat generally increases, and finally the inflammation of the connective tissue about the articulation of the lower jaw makes swallowing almost impossible, and the patient allows the saliva, which is secreted in increased quantity, to run from the mouth. For the same reason the mouth cannot be opened sufficiently to allow of a satisfactory examination except by the finger, which finds the tonsils extremely sensitive to pressure. The swelling of the tonsil interferes with the function of the palate, and the effort at deglutition is liable to be followed by regurgitation of liquids through the nose. The secretion of viscid mucus occasionally produces painful hawking efforts. There is stiffness and soreness on turning the head. The stretching of the posterior pillars by the inflamed tonsil often causes pain referred to the ear. The voice is nasal, and articulation is often painful and very difficult. Respiration is sometimes interfered with if both tonsils are involved. As a rule, however, only one tonsil is attacked at a time, although the second one is apt to pass through the same process after the first has commenced to recover. As the disease progresses the initial constitutional symptoms persist, the fever is often high, and there is diffuse aching throughout the body. If resolution fail to take place, the pain in the tonsils increases, and repeated rigors, occurring after about five or six days, announces the development of suppuration. Finally, the abscess, if not opened by the physician, bursts and immediate relief follows.

Diagnosis.—The disease can scarcely be confounded with any other affection. Phlegmonous pharyngitis limited to the peritonsillar tissue has been already alluded to. There are no differences in the causes or symptoms of the two affections.

Prognosis and Course.—The prognosis is generally good. The majority of cases terminate in resolution, which begins in about five or six days and is finished in two weeks or less, although the process occasionally lasts longer. If both tonsils are simultaneously involved, suffocation may ensue unless treatment give relief. If suppuration occurs, the process generally does not terminate for ten days to two weeks, or even longer. Suppuration increases the gravity of the prognosis, although it is still almost always good. The abscess usually opens internally from the upper part of the gland. The pus has been known to enter the larynx after bursting and to suffocate the patient. Rarely the opening takes place externally near the angle of the jaw. Sometimes the pus burrows down the mediastinum. Penetration of the internal carotid artery, with consequent fatal hæmorrhage, has been reported. Inflammation extending to the larynx may produce fatal œdema of the glottis. Gangrene of the tonsil is an occasional sequel, and permanent hypertrophy of the tonsil may result from repeated attacks of quinsy.

Treatment.—The patient should be confined to bed and placed upon a light diet. If seen early, a saline laxative should be administered and the treatment with either aconite or guaiacum started, each plan of medication having its supporters. Aconite may be given in drop doses of the tincture every half hour or hour. Guaiacum may be given in the form of the ammoniated tincture or as a lozenge, three grains of the resin being taken every two hours. If the case be not seen early, neither of these remedies will have much effect. Effort can still be made to obtain resolution by the constant sucking of ice and the application of an ice-bag externally. Astringent or sedative sprays may also be employed, inasmuch as gargling is often too painful. Inhalation of warm vapors medicated with benzoin, thymol, carbolic acid, and the like is often soothing. The direct application of solutions of various astringents is advocated, among them being nitrate of silver, chloride of zinc, tannic acid, and alum. Scarification, followed by gargling with hot water or by steaming, often gives relief, even if no pus be reached.

When it is probable that an abscess is forming, suppuration should be encouraged by poultices and hot inhalations; the pus should be evacuated as soon as found and antiseptic washes and gargles employed afterward. The internal medication other than that mentioned depends upon the conditions present. Opium may be needed, antipyretics are sometimes called for, and tonics are often required.

CHRONIC HYPERTROPHY OF THE TONSILS, FAUCIAL, PHARYNGEAL, AND LINGUAL.

Definition.—A chronic inflammation producing hypertrophy of the normal tonsillar structures, with impairment of their function.

SYNONYMS.—Chronic tonsillitis; Adenoid growths of the tonsil and the pharynx; Hypertrophy of the lingual tonsil.

Etiology.—In the production of hypertrophy of the faucial tonsils youth is a strongly predisposing factor. The disease is sometimes congenital, and sometimes occurs in babies and young children, but is commoner at from five to twenty years of age. Males are oftener affected than females. Heredity exerts some influence, and syphilis, scrofula, and rachitis act as causes, as do bad hygienic surroundings and repeated digestive disorders. Attacks of diphtheria, scarlet fever, measles, and small-pox are sometimes the exciting causes. Repeated attacks of acute tonsillitis may terminate in chronic tonsillar hypertrophy, and follicular pharyngitis has been said to produce it also. Eczema and impetigenoid eruptions of the face and scalp, and running of the nose, often occur in infants in combination with enlargement of the tonsils. It is uncertain whether the tonsillar condition is the result of other affections, or whether all are produced by one dyscrasic cause. Children sometimes first develop tonsillar hypertrophy at the age of puberty—it is supposed as the result of some sympathetic connection between the tonsils and the genital organs. As a rule, however, the hypertrophied tonsils tend to diminish in size after puberty, and the disease has usually disappeared by the age of thirty years.

Hypertrophy of the pharyngeal tonsil is produced by much the same set of causes, and usually is associated with an overgrowth of the faucial tonsils. Hypertrophy of the lingual tonsil—the adenoid growth at the base of the tongue—is observed principally in adults, is much the commonest in women, and is prone to develop at the menopause. In other respects the causes are the same as act in producing faucial tonsillar hypertrophy.

Pathology.—Both faucial tonsils are generally enlarged, but not always to the same extent. The color of the mucous membrane is pale red, the degree of enlargement varies, and the tonsils may sometimes almost touch in the middle of the throat. All the histological elements of the organs are hypertrophied, but to a varying extent. When the lymphoid elements have especially overgrown, the tonsils are soft, rough, and irregular. Sometimes they present a honeycombed appearance. When the connective tissue has been chiefly affected, they are harder, firmer, and smoother. The lacunæ are always dilated and their walls thickened, and they are frequently filled with and discharge an offensive secretion.

The normal lymphoid tissue of the naso-pharynx presents a firm, smooth, slightly plicated surface. If, however, it has undergone hypertrophy, there are produced masses of papillomatous growth, from the size of a grain of wheat to that of an almond, and sometimes sessile, but usually pedunculated. These are situated on various portions of the mucous membrane lining the naso-pharynx, but especially upon the posterior wall—the position of the pharyngeal tonsil. They are very vascular, reddish, and rather firm. There may be considerable development of adenoid vegetation without much hypertrophy of the faucial tonsils. As a rule, however, the processes go hand in hand.

In hypertrophy of the glandular tissue at the base of the tongue—the lingual tonsil—there is a decided increase in the thickness of that region; the space between the base of the tongue and the epiglottis being filled up by hypertrophied glandular tissue, which gives the parts a somewhat œdematous appearance. One side may be more affected than the other, and sometimes only certain groups of follicles are involved. The blood-vessels are dilated. The movements of the epiglottis are interfered with, and its tip approaches too closely the base of the tongue.

Symptomatology.—It is exceedingly probable that the most important of the signs formerly attributed to enlargement of the tonsils proper depend in most instances chiefly upon the presence of adenoid growths of the pharynx. The symptoms of the two affections cannot well be differentiated in many cases. They are, in fact, identical, except that the obstruction to respiration is more complete in cases of adenoid vegetations, and does not occur in cases of hypertrophy of the tonsils unless the enlargement be very great. The severity of the symptoms in the latter condition depends upon the degree of occlusion caused by the encroachment of the tonsils upon the calibre of the faucial space, and on the degree of pressure which they exert against the soft palate, the tongue, and even the posterior pharyngeal wall.

Although in some instances the disease—whether faucial or pharyngeal, or both—appears to be congenital, and the evidence of obstruction to respiration has been present from the earliest days of life, in most cases the symptoms come on more gradually. The child begins to sleep restlessly, and with the head thrown back and the mouth open, and to snore badly. Sometimes it wakes with sudden attacks of suffocation. It appears to dream as a result of the supplying of the brain with imperfectly aërated blood. Then the habit of keeping the mouth open extends to the day-time as well, owing to the interference with free respiration through the nose, and in well-developed cases the child acquires a peculiar expression and general habitus as a result of this persistent interference. The face looks dull, heavy, stupid, and inane. This is partly due to the constantly open state of the mouth. The lips are thickened, the nostrils small, the superior dental arch is narrowed, and the roof of the mouth elevated. The chest is very commonly deformed, the principal change being a prominence of the upper part of the sternum and a sinking in of the lower portion, with a horizontal circular depression in the thorax corresponding to the attachment of the diaphragm. This is different from the chest of rickets, in which there is a prominence of the whole sternum in addition to the horizontal depression of the thorax referred to. There is also in rickets a vertical flattening of the sides of the thorax, which leaves a large curve behind and a smaller one anteriorly to the costo-chondral articulations, thus producing the "violin-shaped" chest, as it is often called.

The voice is often thick and nasal, and the pronunciation of *l* and *r*, and particularly of *m* and *n*, is impaired. There is increased secretion of pharyngeal mucus. In advanced cases respiration is constantly interfered with, and is easily made labored. The breath is offensive from the hypersecretion of the tonsils. An annoying cough is frequently present, deglutition is somewhat difficult, and there is an uncomfortable sensation in the throat. In infants the enlargement of the tonsils interferes with sucking. Taste and smell are much impaired. Hearing is interfered with, usually by the compression of the Eustachian tube by adenoid growths. The general health is much affected by the deficient oxygenation. The child is anæmic and debilitated and indisposed to exertion.

The appetite is capricious and the digestion often imperfect. Headache is common. Habit chorea of the face has been reported as dependent upon the tonsillar disease. The mental development is distinctly retarded, particularly if the lymphoid structures of the naso-pharynx are much involved, and the child is dull, stupid, and forgetful. The great liability to take cold which results from tonsillar hypertrophy is the cause of frequent sudden exacerbations of the symptoms of obstruction. The existence of tonsillar hypertrophy also increases the liability to diphtheria and the danger in scarlet fever.

The symptoms of hypertrophy of the lingual tonsil are chiefly a disagreeable sensation as of a foreign body, fatigue of the throat in talking or singing, disposition to clear the throat, and occasionally cough and dyspnœa. The patient often refers the sensation to some part much deeper than the base of the tongue.

Diagnosis.—The symptoms described awaken a suspicion of the disease, and direct examination of the parts renders the diagnosis certain. In the cases in which the pillars of the fauces conceal the enlarged tonsils the diagnosis of hypertrophy can be made by manual palpation, with the finger of one hand behind the angle of the jaw and with that of the other upon the tonsil. The diagnosis of adenoid vegetations of the vault of the pharynx can be made with the rhinoscope, or, where this is not practicable, by digital examination. That of the growths at the base of the tongue can be made with the laryngoscope.

Prognosis.—The disease is a very chronic one, and shows no disposition to cease excepting under treatment or with advancing years. Left to themselves, the enlarged faucial and pharyngeal tonsils usually begin to grow smaller by puberty, and the hypertrophy disappears by the age of thirty years or sooner. In the mean time, however, irremediable damage to the patient may have occurred. The liability to this depends altogether on the degree of involvement of the tonsillar organs and upon the age of the patient. If the hypertrophy begin in adult life, it will probably do but little harm. If it occur in infants or young children, and especially if these are already debilitated, the danger of permanent injury is great, although sometimes no evil results follow.

Treatment.—Treatment should be both constitutional and local. The former consists in the carrying out of all measures which will improve the general health, combined with rigid hygienic care so as to avoid as far as possible the occurrence of acute catarrhal attacks. Sufficient clothing, cool bathing, brisk friction of the skin, country air, and digestible food are all important. The patient must be carefully instructed in the proper method of deep diaphragmatic breathing, and of elocution. All efforts at hawking and clearing the throat must be strictly prohibited. Any constitutional disease must receive appropriate treatment. As general alteratives cod-liver oil, iron, quinine, strychnine, or iodide of potassium, with or without minute doses of mercuric chloride, may be employed. Sulphide of calcium, iodoform, and sulphurous waters have been recommended. By internal medication, combined with rigorous hygienic care, the progress of the disease may sometimes be arrested, or, less frequently, retrogression of the hypertrophy be brought about. The latter will only occur if the process be recent and moderate in degree and the tonsils soft.

In nearly all cases local treatment is important. In the milder forms of enlargement of the tonsils, in which the disease is producing no serious general disturbance, various applications can be made. Among the best of these are the compound tincture of iodine; glycerite of tannin; tincture of the chloride of iron and glycerin (1:8); equal parts of powdered alum and tannin rubbed into the glands; nitrate of silver in solution in the strength of 20 grains to the ounce. The latter drug in stick may be applied to the interior of the lacunæ. When the tonsils are much hypertrophied, some treatment is demanded which will destroy the glands. Injections into the body of the tonsil have been recommended. Among the remedies employed for this purpose are ergot, iodine, dilute acetic acid, and carbolic acid. Electrolysis has also been recommended. Applications of London paste once or twice a week to different parts of the surface of the tonsil are highly recommended by Mackenzie. Scarification with the galvano-cautery is also useful, as is the application of chromic acid. These methods of treatment are slow and often unsatisfactory. Consequently, when the general health is being interfered with, and the above measures do not promptly afford some relief, entire or partial removal of the tonsils should be performed. This may be done by the tonsillotome, écraseur, galvano-cautery loop, or the bistoury. The former instrument is the one most generally employed. Free hæmorrhage often follows its use, but is seldom dangerous if precautions be taken not to cut too deeply into the gland.

The treatment of the adenoid growths of the pharynx consists in their removal, and this is even more important than the abscission of the faucial tonsils. Various applications may be made as in the case of those, in the hope of producing absorption or destruction of the mass. It is, however, generally considered much better to remove them at once by operation. This consists in the scraping away of the bodies with the finger-nail or with the curette or forceps. It should be done under ether. A good deal of hæmorrhage is apt to follow, but is easily controlled. The good effects are often almost immediate. Sometimes, however, the bad habits of breathing and speaking will continue for a long time or perhaps permanently.

ULCEROUS TONSILLITIS is included under Ulcerous Pharyngitis.

HERPETIC TONSILLITIS is described under Membranous Pharyngitis.

TONSILLAR CONCRETIONS.

In those who have suffered frequently from acute folliculitis or in cases of chronic tonsillar hypertrophy it is not infrequent to find some of the lacunæ over-distended with secretion, which is greatly increased in amount and altered in character. If the secretion be not too much inspissated, it is sometimes discharged from the tonsil and expectorated in the form of very ill-smelling masses. Sometimes it hardens into actual tonsillar calculi, which may reach the size of a cherry. The softer masses often produce an almost unbearable odor of the breath, while the harder ones may cause some pain in swallowing and may give rise to quinsy.

The treatment consists in that directed to the primary cause and in the removal of the concretions with forceps.

FUNCTIONAL AND SYMPTOMATIC DISEASES OF THE PHARYNX AND TONSILS.

Various diverse conditions may be grouped under this heading.

Anæmia of the Pharynx is symptomatic of general anæmia. The pallor of the palate is especially well marked in phthisis, and sometimes is one of the earliest symptoms of the disease.

Hyperæmia of the Pharynx is the earliest symptom of the various forms of pharyngitis. It is, besides this, seen in the venous congestion of cardiac disease and in that produced by compression of the descending cava from whatever cause. The capillary pulse which attends aortic insufficiency may sometimes be discovered in the pharynx.

Œdema of the Pharynx, especially of the uvula, is sometimes seen in Bright's disease and in great anæmia. Sometimes the uvula becomes very suddenly and greatly enlarged, and may obstruct breathing. Such cases may be the result of an œdematous urticaria attacking the uvula and the veil of the palate.

Hæmorrhage of the Pharynx may result from wounds or inflammatory conditions. Sometimes hæmorrhage may take place into the submucous tissues of various parts of the pharynx.

Anæsthesia of the Pharynx may be general or local, complete or incomplete. It is more frequently seen after diphtheria, although in the adult hysteria is a not infrequent cause. It is also seen in bulbar palsy and in some forms of mental disorder. Its prognosis varies with the cause. It is sometimes an element of danger on account of the liability of food to enter the larynx.

The treatment is that directed to the original disease. Galvanism may also be useful.

Hyperæsthesia of the Pharynx is seen in catarrhal conditions, as already stated. Apart from this, it exists as an idiosyncrasy in many persons, scarcely permitting in them an examination of the throat. In some instances the examination of the throat, or even breathing through the mouth, is actually painful.

Paræsthesia of the Pharynx consists of various altered sensations, as of heat, dryness, pricking, the presence of a foreign body, or the like. It is attended by cough or desire to clear the throat. Examination of the pharynx reveals nothing which justifies the sensation. The affection is often of a purely hysterical nature. It is rarely seen, excepting in states of neurasthenia, chlorosis, hysteria, dyspepsia, or uterine disease, and in those who use the voice a great deal. Sometimes it depends upon varicose veins at the base of the tongue, sometimes it follows the removal of foreign bodies from the throat, and often no cause at all can be found. A very thorough examination of the pharyngeal structures and the exclusion of all other diseased conditions is necessary before a diagnosis can be made. The prognosis is a dubious one. The affection tends to persist very obstinately, but finally to get well. Perseverance in the administration of one of the bromides is the best internal treatment, but it is questionable whether it is advisable to institute it, as it must be continued for so long a time. If the rheumatic diathesis be suspected, the salicylate of sodium or the iodide of potash may be administered. Tonics should be given if indicated. Locally, solutions of antipyrin, menthol, cocoaine, carbolic acid, or the astringents may be tried.

Neuralgia of the Pharynx is a rare affection. Severe darting pain occurs, usually on one side of the posterior wall, whence it radiates to the pillars of the fauces, the nose, the tongue, and sometimes the ear of one side. It grows worse by evening. It occurs in nervous or anæmic individuals, especially women, and may be experienced only at the menstrual period. The attack is sometimes attended by temporary hyperæmia of the pharynx, and sometimes by anæmia of it. The affection may be treated by the use of antipyrin or some allied drug or by the application of tincture of aconite. Quinine may be given if malaria be suspected, or strychnine if there be local hyperæmia. Finally, such constitutional treatment must be employed as the case demands.

Spasm of the Pharynx usually results from severe acute inflammation of the pharynx or from œdema of the uvula. It may, however, be purely nervous, the result of hydrophobia and tetanus, or may also develop in hysteria. The spasm may be tonic, causing great persistent impediment to every effort at deglutition, or it may be clonic, producing a series of elevations of the palate, and consequent opening of the Eustachian tubes, with a resulting snapping sound in the ears.

The diagnosis is to be made by ocular and digital inspection and by the passage of a bougie.

The prognosis varies with the cause. The disease is commonly an affection of long duration, but tends to get well.

Treatment consists first of all in removing the cause when this is possible. The use of the œsophageal bougie, with the administration of bromides and other appropriate remedies, is indicated in hysterical spasm.

Paralyses of the Pharynx.—These are commonly divided into four varieties:

(1) Diphtheritic paralysis is a common affection, developing about two to four weeks after the onset of diphtheria. The soft palate and uvula are relaxed, often to a greater degree on one side. There is difficulty in swallowing, and food, especially liquid, regurgitates into the nose. The voice becomes nasal, and the patients find it impossible to articulate the labial and palatal consonants. Paralysis of the pharynx proper, which is rare, may make swallowing nearly impossible.

The disease may be treated by galvanism and by strychnine given internally, or, better, hypodermically. Feeding with the œsophageal tube is sometimes necessary.

(2) Paralysis of the palate, associated with facial palsy, is sometimes witnessed. The uvula in these cases is drawn to one side, and scarcely moves when the patient breathes. The condition does not demand special treatment.

(3) Paralysis of the tongue and palate is a prominent symptom of bulbar palsy. The affection of the pharynx is shown in difficulty in swallowing, which finally becomes extreme. Nothing can be done in the line of treatment.

(4) Paralysis of the constrictor of the pharynx is associated with paralysis of the œsophageal muscles. It is marked by great dysphagia.

Syphilis of the Pharynx is not infrequently present in various forms: (1) It very often produces a catarrhal pharyngitis, represented by an erythema which attacks principally the palate, pillars, and tonsils. The boundary of these areas is generally very sharply defined. (2) Mucous patches are also a very frequent evidence of secondary syphilis. They are most commonly found on the soft palate and on the pillars. They produce some soreness, especially on swallowing. (3) Ulcers of various forms may be present. The initial primary sore is occasionally seen on one of the tonsils. Tertiary ulcers, either superficial or the result of gummata, are more often found. They are usually seen on the posterior wall of the pharynx, the palate, and the pillars. The damage done by them may be very extensive.

The treatment of syphilitic lesions is primarily that of the disease itself. Ulcers may be treated locally with solutions of sulphate of zinc or sulphate of copper; or occasional light applications of nitric acid may be made, with repeated applications of iodoform in the intervals.

Tuberculosis of the Pharynx may rarely show itself in the form of a primary acute tubercular pharyngitis. This occurs in persons who have either simultaneously developed pulmonary phthisis or perhaps suffered first from a pharyngeal involvement. Very numerous grayish tubercles, which bleed easily, appear in patches on various portions of the mucous membrane of the pharyngeal structures. They soon form irregular superficial ulcers, which have indistinct edges and which coalesce to some extent. Fresh tubercles go on forming, which very soon break down into new ulcers. The principal symptoms are unusually great pain on swallowing, cough, high fever, increasing debility, and emaciation. Dyspnœa resulting from the pulmonary complication usually attends. The disease is most apt to be confounded with syphilis, from which it is distinguished by the great pain and the presence of tubercles around the ulcers, and the evidence of pulmonary involvement.

The prognosis is very unfavorable, and little can be done in the way of treatment. Hot inhalations and sedative applications are of value to relieve pain, and insufflation of morphine or iodoform may be employed for the same purpose.

Besides this rare primary form, secondary tuberculosis of the pharynx and tonsils shows itself by the ulceration which quite often develops in the later stages of advanced phthisis. These ulcers, like those described, are very painful.

Certain of the Infectious Fevers exert their influences upon the pharynx.

Typhoid fever occasionally produces ulcers in the pharynx, probably of the same nature as those of the intestine. The eruptions of variola, rubeola, rubella, and scarlatina have already been referred to in various places, particularly in discussing the affections of the mouth. Erysipelas of the throat has likewise been already referred to. It may produce severe catarrhal pharyngitis, or it may be attended by the production of vesicles which change into ulcers, or it may terminate in gangrene.

Malignant and Non-malignant New Growths of the pharynx and tonsils may be referred to in this connection, although they are not often met with. Cancer seldom attacks any but the lowest portion of the pharynx close above the œsophagus. It is very liable to spread to the larynx. The symptoms are nearly identical with those of cancer of the upper portion of the œsophagus. If the cancer be in the pharyngo-oral cavity, there is much pain on swallowing and speech is interfered with. Cancer occasionally, but very rarely, attacks the tonsils. Sarcomata, osteomata, cystomata, papillomata, and other growths have all occurred in the pharyngeal structures. Lymphomata of the tonsils have been one of the first evidences of lymphatic anæmia. Any further discussion of the subject is rather in the domain of surgery.

DISEASES OF THE ŒSOPHAGUS.

By WILLIAM PEPPER.

ŒSOPHAGITIS.

ŒSOPHAGITIS may be divided in the same manner as pharyngitis, according to the nature of the inflammatory process, whether catarrhal, phlegmonous, and so on. So, also, it may be spoken of as acute or chronic, or it may be subdivided, according to the cause, into idiopathic, deuteropathic, or traumatic. Inasmuch as the disease is not very common, and as the differences of the individual varieties have no especial clinical interest, it is more expedient to consider the subject under one heading.

Definition.—An inflammation of the œsophagus, diffuse or circumscribed, and either superficial or more deeply situated and with destruction of tissue.

Etiology.—*Acute Catarrhal Œsophagitis* may result from exposure to cold, extension of the catarrh from the pharynx or the stomach, the existence of a specific fever, the presence of a rheumatic diathesis, the ingestion of alcohol, the use of food or drink which is too hot or too cold, the swallowing of hard substances, caustics, or other irritant fluids in not too great concentration, the administration of certain drugs, especially of mercury, and the passage of a bougie. It has also been known to arise spontaneously in sucklings.

Chronic Catarrhal Œsophagitis may follow the acute process through the prolonged action of the causes which produce it. It may result from the abuse of tobacco, or especially of alcohol, the pressure from without of tumors, enlarged glands, or an aneurism, and the presence of disease of the œsophagus itself, as carcinoma, dilatation, and the like. It is also encountered in association with syphilis and phthisis, and as a result of heart disease and of cirrhosis of the liver.

Mycotic Œsophagitis may result from the extension of thrush to the œsophagus.

Pseudo-membranous Œsophagitis may occur especially in diphtheria of the pharynx, but occasionally in pneumonia, dysentery, cholera, carcinoma, pyæmia, nephritis, tuberculosis, and the eruptive fevers. It may also rarely develop in otherwise perfectly healthy persons, as in a case reported by Reichman.

Phlegmonous Œsophagitis may arise as a sequel to the catarrhal or pseudo-membranous form, being brought about by the same causes which are active in their production. It may also result from the swallowing of foreign bodies, or more especially the sticking of these in the œsophagus, from suppuration in the neighborhood of the tube, as from tubercular lymphatic glands or caries of the vertebræ, or from laryngeal perichondritis. Often the affection seems to arise idiopathically and the cause cannot be discovered.

Corrosive Œsophagitis, often with a secondary involvement of the submucous connective tissue, results from the ingestion of powerfully caustic substances.

A *Pustular Œsophagitis* occurs in small-pox, and sometimes after large doses of antimony.

Ulceration of the Œsophagus may follow in the course of œsophageal inflammation. A catarrhal process, whether acute or chronic, may cause superficial erosions or follicular ulcers, as may also the pseudo-membranous inflammation. Powerful corrosive liquids and impacted foreign bodies occasion more or less death of the mucous membrane, with ulceration and sloughing. Cancer produces ulceration, and pressure from the outside may do the same. The round or peptic ulcer of the œsophagus, similar in nature and cause to the round ulcer of the stomach, may be neurotrophic in origin, or may possibly be produced by the action of the gastric secretion which has entered the œsophagus during vomiting.

Pathology.—In the acute catarrhal variety the lesions may be either diffuse or circumscribed. The mucous membrane is thickened and softened, but, as seen at autopsies, is generally not red. Patches of epithelium are seen to have desquamated, and in one case reported by Birch-Hirschfeld a membranous tube, composed of the epithelial lining, was vomited during life. Numerous minute erosions are sometimes visible and distinct ulceration is occasionally seen. Often the mucous follicles are decidedly hypertrophied (follicular œsophagitis).

In chronic œsophagitis the mucous membrane is of a grayish-red or brownish-red color, much thickened, covered with muco-purulent secretion, and sometimes polypoid in appearance. The œsophageal veins in the neighborhood of the stomach are sometimes greatly distended. In the pseudomembranous variety there is the characteristic exudation over different parts of the œsophageal lining, but most prominently at the upper portion of the tube. Ulceration sometimes forms beneath the pseudo-membrane.

Mycotic œsophagitis exhibits the characteristic growth of thrush, as seen in the mouth. The fungus may cover a large portion of the œsophagus, and even occlude it entirely. It is most abundant near the cardiac end. In phlegmonous œsophagitis there is an infiltration of the submucous connective tissue with pus, which pushes the mucous membrane before it toward the interior of the tube. The membrane itself is congested, partially denuded of epithelium, covered with mucus, and may be ulcerated in places. Gangrene may result. The process may be diffuse or circumscribed. The action of strong corrosive poisons first involves the mucous membrane, and thence extends to the submucous connective tissue. The mucous membrane presents a grayish or blackish discoloration, and is later more or less detached as a slough, leaving ulcers which form dense scar-tissue if healing takes place.

Symptomatology and Diagnosis.—In the milder cases no symptoms are apparent, but in instances of decided inflammation of the œsophagus pain on deglutition is the principal evidence of the affection. Sometimes there is, in addition, a constant dull pain beneath the sternum, between the shoulders, or in the neck. The position of the pain does not always correspond to the seat of the inflammation. It is sometimes increased by moving the head or the spine.

There may be difficulty in deglutition, which may depend upon a reflex spasm produced by the great pain attending efforts at swallowing or upon a narrowing of the lumen of the œsophagus through swelling of the mucous membrane, or which may be the result of paralysis of the muscular walls through inflammatory products. Portions of the food taken may be regurgitated with mucus, and sometimes with blood. The presence of blood and pus indicates ulceration which may depend on the presence of a foreign body, upon phlegmonous œsophagitis from other cause, or upon the ulceration produced by poisons. Considerable febrile reaction also attends the phlegmonous form of the disease. The recognition of pseudo-membranous œsophagitis can only be accomplished by finding membrane which had been vomited or by discovering symptoms of œsophagitis in the course of pneumonia or of membranous pharyngitis. Thrush of the œsophagus may produce no symptoms or may lead to entire inability to swallow as the result of obstruction.

The diagnosis of inflammation from other diseases of the œsophagus will be referred to in considering the latter.

Prognosis and Course.—The prognosis of acute catarrhal œsophagus is good, the symptoms, if any, lasting a few days at most. Sometimes, however, the disease becomes chronic. In corrosive œsophagitis the prognosis is grave. Even if the patient recover from the action of a corrosive poison, stricture is almost certain to develop after some weeks or months. Phlegmonous œsophagitis is also a very severe disease, and may result fatally in three to four days. It may give rise to much prolonged suppuration, and yet end in recovery. It sometimes terminates in gangrene. Ulcers of the œsophagus may perforate into surrounding structures, and are very liable to prove fatal. Recovery from the phlegmonous form is prone to leave cicatricial contractions. Chronic œsophagitis may finally produce dilatation of the œsophagus. Pseudo-membranous inflammation of the œsophagus is of bad augury.

Treatment.—The milder forms of œsophagitis require no treatment other than rest in the house and demulcent drinks. Ice is pleasant to some patients, and may be sucked to quench thirst. In severer cases it is sometimes necessary to resort to nutrient enemata and to avoid swallowing as far as possible. Injections of morphine may be needed. In chronic catarrhal pharyngitis the primary cause must be treated if it can be found. Locally, astringent or alterant solutions or ointments, such as those of tannic acid, alum, nitrate of silver, and iodine, may be applied on a soft sponge attached to a sound, or on a flexible bougie. This treatment should not be used if the patient suffer any pain from it. It is generally best to avoid the use of the sound in any form of œsophagitis unless there is prospect of some very positive good results being gained by it.

Stenosis of the Œsophagus.

Definition.—A narrowing of the calibre of the œsophagus, produced by organic changes in the wall of the tube, by pressure from without, or by abnormal intra-œsophageal conditions.

Etiology.—Various causes produce this, the commonest of œsophageal disorders. Among the causes of intra-œsophageal stenosis are to be mentioned the presence of foreign bodies which have been swallowed and have remained fast in some portion; polypoid tumors projecting into the lumen; and the presence of large masses of the thrush fungus.

Among interstitial causes, in which the stenosis is due to disease of the wall of the tube, carcinoma is to be mentioned as by far the most frequent. Another common cause is cicatricial contraction of the wall of the œsophagus, the result of the healing of ulceration of any sort, especially that following the action of corrosive substances. Among other similarly acting, but less frequent causes, are wounds of any kind, syphilitic and tubercular ulcers, the ulceration of the pustular œsophagitis of small-pox, and the ulcus œsophagi rotundum. Occasionally a congenital constriction of the lumen of the œsophagus is observed. A chronic œsophagitis may finally lead to great thickening of the mucous lining, with hyperplasia and infiltration of the submucous tissue, and consequent stenosis. Abscesses in the wall of the œsophagus may occlude by pressure. Spasm of the œsophagus is another cause of stenosis.

Among the causes of narrowing of the lumen of the œsophagus by compression from without the tube are enlargements of the thyroid gland, tumors, enlarged lymphatic glands, abscess of the vertebræ, and aneurism.

Pathology.—This varies largely with the cause. The stenosis may occur anywhere, but is most frequently situated in the lower third, and next oftenest near the bifurcation of the trachea. The stricture is usually single. The proportion of the length and of the circumference of the œsophagus affected, and the degree of encroachment upon its lumen, vary within wide limits. The obstruction may be almost complete. Sometimes nearly the entire length of the œsophagus is involved, but this is rare. Dilatation of the portion above the stricture is apt to develop.

Symptomatology.—The chief symptom of stenosis of the œsophagus is dysphagia. This may come on suddenly or slowly, according to the nature of the cause. The slow onset is the most common. In it there is first noticed difficulty in swallowing large masses of food, and the patient finds it necessary to help the act by at once drinking some liquid. The sensation of the sticking of food is referred to different portions of the body between the mouth and the stomach, and but little dependence can be placed upon this. Later, as the disease advances, it requires repeated efforts to swallow any solid food, and the attempt is followed by pain and often by regurgitation. Often the first bolus causes the greatest pain. The patient sometimes finds it necessary to manipulate the side of the neck or to make certain turning movements of the head in the effort to aid the passage of the bolus. Sometimes the food lodged above the stricture presses upon the trachea and causes suffocative attacks. Finally, it often happens that solid food cannot be swallowed at all.

If the stricture be high up, regurgitation takes place immediately. If it be more deeply placed, some dilatation of the tube is apt to follow, and the food consequently often remains in the œsophagus for hours, and is then regurgitated in a softened condition and mixed with mucus. Its reaction is alkaline, which shows that it has not reached the stomach. It often happens in very bad cases that even fluid cannot be swallowed except in very small quantities.

Diagnosis.—The diagnosis of the disease can generally be readily made from the symptoms already detailed. It may be aided by auscultation and by the use of a sound. If the œsophagus be ausculted along the left side of the trachea as far as the clavicle, and then along the left side of the vertebral column, and the patient meanwhile swallow a mouthful of water, a gurgling may be at once heard as far as the seat of the stricture. Below this it is entirely absent or only audible after a pause. Later a variety of noises may be heard in the part above the obstruction.

The employment of the œsophageal sound is a more exact method of determining the position and degree of the obstruction. Either the olive-tipped bougie or the elastic stomach-tube can be used. The patient should sit upright with the head thrown back. The operator should then pass the sound along the first and second fingers of the left hand, inserted in the mouth, deeply backward to the posterior wall of the pharynx. Slight resistance is normally encountered as the instrument passes the cricoid cartilage. The employment of the sound must be made very carefully and without any undue force, since fatal accidents may occur from the perforation of an ulcer. It is necessary to determine whether an aneurism is present before the sound is used, since its existence absolutely prohibits this means of diagnosis.

The determination of the nature of the stenosis in different cases may be largely aided by the previous history of the case, a history of the ingestion of corrosive substances indicating that the lesion is cicatricial, while a slowly-advancing obstruction in elderly persons is almost always due to carcinoma.

The diagnosis of functional from organic stenosis will be considered under Spasm of the Œsophagus.

Prognosis and Course.—The prognosis depends largely upon the cause. When the disease is due to cancer it is absolutely unfavorable. Cases of this nature often show temporary improvement, the result of the breaking down of a portion of the obstructing mass. Aneurisms leave no hope of improvement in the symptoms.

The course of incurable cases depends altogether upon the degree of stenosis. When it is extreme, all the signs of great and increasing inanition develop, and death follows from starvation. In other cases a fatal termination is the result of perforation of an ulcer into surrounding parts. In cases due to pressure of a submucous suppuration sudden relief may follow the bursting of the abscess into the œsophagus. The prognosis in cicatricial contraction is more favorable both as to length of life and as to ultimate recovery under treatment. Without treatment, however, the stenosis tends to get worse, and finally the patient dies of starvation.

Treatment.—The cause must be treated as far as possible, although, as a rule, little can be done in this line. All possible measures to maintain the strength are indicated, and the patient must be fed by the rectum if sufficient nourishment cannot be given by the mouth. In cicatricial stenosis much may often be gained by mechanical treatment. This consists in systematic gradual dilatation of the stricture. It must be persisted in for a long time, even after great improvement has occurred, lest the contraction return. The advisability of dilatation in the case of carcinoma is questionable. Sometimes very good though temporary results are obtained, but there is some danger of perforation. It is often advisable to combine dilatation with feeding through a stomach-tube. Electrolysis has been highly recommended, and has been undoubtedly useful in some cases. Where dilatation cannot be successfully employed, and the stenosis is extreme, œsophagotomy or gastrotomy may be indicated.

DILATATION OF THE ŒSOPHAGUS.

Definition.—A distention of all or of a portion of the œsophagus, either in its entire circumference or in part of it.

SYNONYMS.—Ectasia of the œsophagus; Diverticula of the œsophagus; Hernia of the œsophagus; Œsophagocele.

Etiology.—A distention which affects the entire circumference of the tube is called a *dilatation*. If only a portion of it be involved, so that a pouch is formed, the condition is called a *diverticulum*.

A diffuse dilatation of the whole œsophagus is very commonly the result of obstruction at the cardiac end, although sometimes the result of paralysis and sometimes a congenital affection without discoverable cause. Annular dilatation is sometimes congenital and sometimes the result of stricture.

Diverticulum, or pouched dilatation, may be of two forms, *pulsion* and *traction*. The first, a very rare disease, is the result of pressure from inside the œsophagus. It has been found most frequently in elderly men. In a recent case, the symptoms had lasted with gradually increasing severity for at least ten years. It generally results from retention of food just above a foreign body. This latter, or injury received in some other way, has damaged the muscular coat and allowed the formation of a pouch. Once started, every portion of food which presses on it increases its size.

The traction diverticulum is more frequent. It is seen at any age, and oftener in scrofulous persons. It arises from a contracting process, especially of the bronchial glands, taking place in the neighborhood of the œsophagus. A periadenitis attaches an enlarged bronchial gland to the outside of the œsophagus, and the gland, then contracting through a degenerative process, pulls with it a funnel-formed diverticulum.

Pathology.—A diffuse dilatation is generally spindle-shaped, larger at the bottom and growing smaller above. The evidences of chronic œsophagitis with erosions and ulceration are often present, and the muscular coat is much hypertrophied. Sometimes, however, the muscular layer is very thin. The dilatation occasionally reaches a remarkable size.

Annular dilatation is situated either just above a stricture or immediately above the diaphragm. It is largest at its upper portion. The muscular coat is generally hypertrophied.

In the pulsion diverticulum the muscular coat has given way, and the mucous and submucous layers have projected, hernia-like, through this opening. The mucous membrane is generally chronically inflamed. The size of the pouch may vary from very small dimensions up to even four inches or more in length. Its situation is nearly always on the posterior wall of the highest part of the œsophagus, at its juncture with the pharynx.

The traction diverticulum may or may not be covered by the muscular layer. It is usually situated above the position of the bifurcation of the trachea, occupies the anterior or lateral portion of the œsophagus, and is seldom more than about one-quarter of an inch deep. Occasionally two or three diverticula are present in the same case.

Symptomatology and Diagnosis.—The symptoms of large diffuse dilatations are largely those of stenosis. In addition to this, the presence of dilatation is probable when regurgitation does not take place for several hours after eating. The patient is often conscious that the food has not reached the stomach. Sometimes the large amount of food in the œsophagus produces pressure-symptoms, such as palpitation, oppression, and threatened suffocation. If the œsophagus be artificially distended with carbonic dioxide, an unusually tympanitic region may sometimes be discovered along the vertebral column. On the other hand, if the tube be full of food this region is dull on percussion. The sound passed into the œsophagus sometimes permits of an unusual degree of lateral movement. If it passes easily into the stomach, it is proof that the dilatation is a congenital or a paralytic one, and not the result of stenosis.

In annular dilatation the food is apt to be regurgitated more quickly, but here, too, it is sometimes long retained. If the dilatation be high in the neck, it may usually be palpated there when filled with food, disappearing when the food is regurgitated.

The pulsion diverticulum is without symptoms at first. As it becomes larger, it produces some distress in the neck, and a tumor there which can often be emptied by pressure and whose size varies at different times. Peculiar clicking and splashing noises are often audible in it while the patient is eating. The contents of the diverticulum often decompose and produce a foul breath. As the diverticulum grows in size the danger increases, for it finally compresses the œsophagus completely, and every effort to eat only makes the pouch fuller. Only after prolonged efforts at retching and vomiting is the sac emptied and entrance of food into the stomach possible. The diagnosis of a diverticulum is made very probable if the sound sometimes is passed easily into the stomach and sometimes catches high in the œsophagus.

The traction diverticulum presents no symptoms.

Prognosis and Course.—The prognosis of dilatation and of pulsion diverticulum is unfavorable unless the cause can be removed; and even with its removal recovery cannot be ensured. Death from inanition is the usual termination after a more or less tedious course. Sometimes death results from perforation of an ulcer. The only danger attending a traction diverticulum is that of ulceration and perforation.

Treatment.—The treatment must consist principally of efforts to remove the cause and to sustain the patient's strength in the methods already described in discussing Stenosis.

CANCER OF THE ŒSOPHAGUS.

Carcinoma, the most frequent disease of the œsophagus, is nearly always primary, though in rare cases it occurs as a secondary extension, particularly from the stomach. Males are oftener attacked, and generally after the age of forty years. Possibly local injury from any source, as by hot and corrosive liquids, the action of hard foreign bodies, or the excessive use of alcohol, may have some agency in its production, but this is still in doubt.

The growth is nearly always a squamous epithelioma, which is oftenest situated in the lower third of the œsophagus. The three favorite points of election appear to be just above the cardia, the position of the crossing of the left bronchus, and the region behind the cricoid cartilage. The growth begins in the mucous membrane, soon implicates the entire thickness of the wall of the œsophagus, extends around its circumference like a ring, and speedily ulcerates. In the direction of the length of the œsophagus it does not often exceed four inches, although occasionally involving the greater part of the tube. Stenosis results, with dilatation of the œsophagus above it and hypertrophy of its walls. As the disease advances it may extend to the neighboring tissues, as the stomach, pharynx, lymphatic glands, trachea, bronchi, mediastinum, lungs, heart, aorta, and vertebræ.. Perforation of an ulcer may likewise take place into them. Spread of the disease by metastasis is less common.

The symptoms of cancer are those of a slowly-developing stenosis, as already described. Sometimes evidence of cachexia comes on before the symptoms of obstruction appear. Rarely there are at no time local evidences of œsophageal carcinoma, and the patient dies of the cachexia or of complications. A symptom characteristic of carcinoma of the œsophagus is the temporary marked relief of the stenosis which sometimes follows the breaking down of obstructing ulcerating masses. Various secondary involvements of neighboring tissues present corresponding secondary symptoms. One of these involvements not infrequently observed is gangrene of the lung following perforation of an ulcer into the trachea or bronchi, or the result of aspiration of vomited decomposing material. Not rarely, too, the recurrent laryngeal nerve is involved by the growth, and alteration of the voice is produced.

The diagnosis of cancer of the œsophagus is to be made by establishing first the existence of organic stenosis as apart from spasmodic stricture; and subsequently by a consideration of the history of the case, the age of the patient, and by the existence of cachexia.

The prognosis of œsophageal cancer is hopeless, although patients may live for months or rarely even for one to one and a half years or longer. Death usually takes place from starvation or from cachexia, but sometimes from the results of a perforation into neighboring parts.

The treatment has already been discussed under the heading of Stenosis.

RUPTURE OF THE ŒSOPHAGUS.

The sudden rupture of a previously healthy œsophagus is of very rare occurrence, and only a few cases have been reported. It has usually taken place in men and during vomiting in an intoxicated state or after a full meal. Zenker has claimed that it is the result of œsophagomalacia occurring during life. The lesion consists of a tear, usually longitudinal, through the entire œsophageal wall. It is generally situated near the cardia. Portions of food pass through it into the surrounding parts, generally the mediastinum and thence into the pleural cavities.

The symptoms come on with the greatest suddenness. There is intense pain referred to the interscapular region, and all the evidences of collapse. Cutaneous emphysema usually develops in the neck and chest. The disease may be suspected if, with these symptoms, there occur vomiting of blood, evidences of pleural effusion, and pneumothorax. It would be necessary, however, to exclude previous œsophageal disease and perforation of the stomach.

The prognosis is absolutely unfavorable. Death generally occurs in a few hours, but sometimes not for several days.

The treatment is entirely symptomatic. No medicine or food should be given by the mouth.

ŒSOPHAGOMALACIA.

Softening of the walls of the œsophagus is in the vast majority of cases a post-mortem lesion. Very rarely it may develop when the patient is moribund, especially in diseases of the brain. In other very exceptional cases it may possibly develop during apparent health, and be the predisposing cause of rupture of the œsophagus. This has been referred to in discussing the latter affection. It is often associated with gastromalacia. The direct cause is the digestive action of the gastric juice which has made its way into the œsophagus.

The posterior part of the lower segment of the tube is the region nearly invariably involved. The mucous membrane is softened, gelatinous in appearance, and of a color varying from yellowish-brown to greenish-black. If the affection penetrate more deeply, the submucous and muscular layers are likewise softened, gelatinous, and discolored. Rupture is liable finally to take place. Hæmorrhagic infarction has been seen in the portion of the walls adjacent to the affected spot. This is a proof that the softening took place during life.

The diagnosis of softening during life could only be made when rupture had taken place, and the symptoms then are those already described.

PARALYSIS OF THE ŒSOPHAGUS.

Very little positive is known of this rare affection. It may develop in disease of the brain and cervical cord. Probably the paralysis which involves the pharynx in bulbar palsy may extend to the œsophagus as well. Diphtheria appears occasionally to produce it. It may also attend hysteria or follow pressure of enlarged lymphatic glands upon or other injury of the pneumogastric nerves. Among other causes which have been assigned are syphilis, alcohol, lead, and exposure to heat and cold.

The symptoms consist of difficulty in swallowing, although examination with the sound reveals no stricture. Finally, the œsophagus may become dilated and the symptoms of paralytic dilatation develop as already described.

The prognosis depends upon the ability to remove the cause.

Treatment consists in nourishing the patient, if necessary, with a stomach-tube. Cautious faradization of the interior of the œsophagus and remedies directed to the cause of the affection may also be tried.

SPASM OF THE ŒSOPHAGUS.

Definition.—A partial or complete spasmodic contraction of the muscles of the œsophagus.

SYNONYMS.—Spasmodic stricture of the œsophagus; Œsophagismus; Cramp of the œsophagus.

Etiology.—The affection is much the commonest in females of early adult age. Epilepsy, chorea, hysteria, tetanus, and hypochondriasis may produce it. Hydrophobia is a prominent cause, and psychic conditions, as fright or joy, may excite it. It is, for example, sometimes seen in those who imagine they have been bitten by a rabid dog. The mere dread of strangling in those who have experienced the sensation is sufficient to bring on another attack. It may be associated with neuroses. The affection may arise reflexly from pregnancy, lactation, or from diseases of various distant organs, among them the pharynx, stomach, intestines, heart, lungs, brain, and cord, and especially the female genital organs. Rheumatism and gout are said sometimes to cause it. Occasionally spasm is excited by various organic diseases of the œsophagus.

Symptomatology.—The attack comes on suddenly, and is accompanied by sensations of constriction in the throat or chest, which may be painful. Palpitation, hiccough, oppression, syncope, or even general convulsions, may also develop. The spasm may attend every effort at swallowing, or may occur only at irregular intervals. Sometimes the very thought of swallowing will bring on an attack. It may be painful or painless, and may last for hours or for days, or even for months. It may be induced only by certain kinds of food. Solid food is generally that with which the greatest difficulty is experienced. It is only rarely that the patient is entirely unable to swallow liquids as well. If the constriction be high in the œsophagus, food may be regurgitated at once. If lower, it may be retained for a longer time and then ejected, or if the spasm soon relaxes it finally passes into the stomach.

Examination with the sound reveals a resistance which soon yields, or may discover no narrowing whatever. The seat of spasm is oftenest either near the upper end of the œsophagus or near the cardia, but it does not always remain the same in any one case. Auscultation also shows the position of the stricture.

Diagnosis.—The suddenness of onset, the intermittence, the coexistence of nervous causes or other nervous symptoms, and the examination with the sound usually make the diagnosis clear. The general symptoms and the history of the case deserve careful attention owing to the great importance of distinguishing spasmodic stricture from the early stage of organic disease of the œsophagus. When the patient is a young neurotic female and the symptoms of obstruction, although urgent, are intermittent and the general health suffers but little, the diagnosis may be made even without the passage of a sound. When, however, the subject is a male adult, and when, as I have seen, the spasm is so persistent and extreme that swallowing is rendered impossible so that emaciation advances rapidly, the use of the sound becomes essential.

Prognosis and Course.—These are nearly always favorable, except where the cause is irremediable and itself an element of danger. Even if the trouble be very persistent, enough food can usually be taken in some form to prevent material loss of health. In the rare cases where great emaciation is caused, remarkable recovery will follow the use of the sound combined with proper treatment, if the spasm is not associated with some grave condition.

The duration of the disease is very variable, and depends upon the nature of the cause and the ability to remove it. There is a great tendency to recur shown in purely neurotic cases.

Treatment.—Constitutional treatment must be directed against the general neurotic condition which so often attends. The valerianates, asafœtida, the bromides, and sedative remedies in general are indicated. Local treatment is nearly always required. Occasionally a single passage of the sound is followed by a complete arrest of the tendency to spasm, and I have seen marvellously rapid recovery of flesh and strength thus effected. Persistence in the use of the sound is, however, more commonly required to secure a permanent cure. It is frequently well to use the sound just before food is taken. The patient must be made to swallow food in the presence of a physician or nurse, in order that the fear of its sticking in the throat may be allayed by the confidence this presence inspires. Feeding through the tube is required in some cases if nutrition seem to be in danger of suffering. Very careful faradization of the interior of the œsophagus may be tried, bearing in mind the danger from the close proximity of the pneumogastric nerve.

DISEASES OF THE STOMACH.

BY WILLIAM PEPPER.

GENERAL CONSIDERATIONS.

Position and Size of the Stomach.—The stomach normally occupies chiefly the left hypochondrium, the epigastrium, and part of the right hypochondrium. It is situated obliquely, with its axis more nearly vertical than horizontal. Much the greater part of it lies to the left of the median line, and only about a quarter or less of it is to the right side. It is in relation above with the liver and diaphragm; below, with the transverse colon; in front, with the abdominal wall, the diaphragm, and the liver; behind, with the pancreas, the crura of the diaphragm, the solar plexus, and the great abdominal vessels. It touches the spleen on the left and the gall-bladder on the right. The cardiac orifice lies behind the left seventh costal cartilage near the sternum, and is covered by the liver. The pyloric orifice is on the level of the inner end of the right eighth costal cartilage, and close to a vertical line passing along the right edge of the sternum, but its position shifts considerably according as the stomach is full or empty. It is covered by the left lobe of the liver. The fundus reaches upward as far as the fifth rib on the left side and downward as far as one to two and a half inches above the navel—*i. e.* about midway between it and the tip of the ensiform cartilage. The position of the lower border, however, is capable of great variation. About two-thirds of the stomach lie in the left hypochondrium under the ribs, and cannot be reached by palpation. The lesser curvature also is out of reach, being covered by the left lobe of the liver. Through most of its course it is nearly parallel with the spinal column and to its left. The greatest vertical diameter of the normal gastric resonance is stated by Pacanowski to vary from about four to five and a half inches. The actual capacity of the normal stomach is very variable and difficult of determination. A series of measurements reported by Ewald showed a range of from eight to fifty-six fluidounces. The normal shape of the stomach, too, may vary considerably without approaching what can be called an abnormal condition.

Classification of Gastric Diseases.—The studies upon the diseases of the stomach which have been prosecuted with so much vigor and success during recent years have necessitated a considerable alteration in their classification as compared with that found in the older text-books. However convenient it was from a clinical standpoint to speak of atonic dyspepsia, acute dyspepsia, flatulent dyspepsia, nervous dyspepsia, gastric catarrh, and the like as always separate and distinct entities, to follow this course at the present time would be to disregard the now well-determined pathological conditions present in the different gastric disorders. For instance, the term "catarrhal" cannot be properly applied to the mucous membrane of the stomach, inasmuch as nearly every condition commonly called by this name involves a disturbance of the gastric glands, and not merely of the surface of the mucous membrane, and is consequently a parenchymatous disorder. It is only when the orifices of the glands, *i. e.*, the surface of the mucous membranes, are alone involved that the term catarrhal can be properly used, and such a condition must be very unusual, owing to the tendency to more extensive pathological changes which even mild cases undergo.

The subject of classification is, indeed, an exceptionally difficult one. There certainly are numerous cases in which mere functional disturbance largely predominates, and some in which the whole condition would appear to be dependent upon a nervous disorder. At the other end of the scale are cases in which there is beyond doubt extensive inflammatory alteration of the mucous membrane. Between these extremes there are disturbances of all degrees. The existence of gastric neuroses, including purely functional, non-inflammatory affections, must be admitted; but in addition to these there are the inflammatory gastric diseases, which, however, are always accompanied by disturbance of function. It is often difficult or impossible to determine from the gastric symptoms alone which cases are functional only and which have some inflammatory change as a basis or accompaniment. The various attendant symptoms connected with other parts of the body, the condition of the general health, and the like, may need consideration in order to throw light upon this point.

It is evident, therefore, that any classification adopted must be allowed a certain latitude. Nevertheless, the more the subject is closely studied, the more frequently does an element of inflammatory change show itself to be present in some degree, and the number of cases of purely nervous disturbance is proportionately reduced, although some still remain undoubted members of the class.

Under *gastritis*, either acute or chronic, may then be classified all those cases in which it is probable that some inflammatory change is present; including here all those cases commonly spoken of as gastric catarrh. Under *neuroses* of the stomach may be placed the cases of simply nervous disease, whether of the nature of a dyspepsia or of other sort. Cancer, ulcer, and some other conditions must receive separate consideration.

Physical Methods of Examination of the Stomach.—Without any special mode of procedure the position and size of the stomach can sometimes be partially determined by percussion. The liver, however, covers a part of the right boundary, and the left boundary also cannot be satisfactorily determined. The upper border is partly covered by lung. The lower boundary—which, after all, is the important one—can often be located by the difference in the percussion-notes of the stomach and the adjacent colon and small intestines. Frequently, however, percussion done in this manner is unsuccessful.

The development of a succussion splash by striking the stomach with the tips of the fingers of one hand has been recommended as a reliable means of outlining the organ. It is, however, often unsatisfactory. Palpation should be practised with the hand placed horizontally, and the pressure should be gentle and gradually increased, in order to avoid producing contraction of the muscles or pain. Sometimes the knee-elbow position will bring to light conditions not discoverable when the patient is upon the back.

To aid ordinary physical examination special methods have been recommended. Leube advocated passing a stiff sound into the stomach and feeling for its end through the abdominal walls. The method is not unattended by danger if ulcer be present, and is not to be recommended for various other reasons. The end of the tube can only be felt if the abdominal walls be relaxed; it does not always reach the lowest portion, but may impinge upon some other part of the wall of the stomach; and, finally, in cases of vertical position of the stomach, the position of the tip of the sound leads to fallacious conclusions.

Distention of the stomach with carbonic dioxide, first advocated by Frerichs, is a common and favorite method of determining the gastric boundaries. The patient should drink about one teaspoonful of bicarbonate of sodium dissolved in a little water, and immediately after this the same amount of tartaric or other vegetable acid dissolved in water sweetened with a little sugar. The distention of the viscus which ensues often renders the boundary distinct both to inspection and to percussion. A stomach-tube should always be at hand with which to remove the gas in case symptoms of oppression develop. In place of carbonic dioxide air may be pumped into the stomach through a stomach-tube connected with a hand-bulb apparatus. This has the advantage that the amount of air introduced can be accurately registered.

The method proposed by Piorry consists in allowing the patient to drink, or administering to him through a tube, several glasses of water, and then determining the lower border of the percussion dulness while he is in the erect position. This dulness, which shifts when he again becomes recumbent, represents the lower boundary of the stomach. Dehio modified this by introducing about one quart of water in divided amounts of about a half pint each, and by determining the lower limit of the dull area after each ingestion.

These methods are not always conclusive, since loops of intestine may lie in front of the stomach and interfere with the percussion; and it is, further, not always easy to distinguish between colon and stomach even when this interference does not occur. It is, therefore, sometimes well to fill the stomach entirely with fluid, and then to contrast its dulness with the tympany of the colon; and, to help in the matter, air may be pumped into the colon from the rectum, and its percussion note altered in pitch in this manner. The reverse may be done and the stomach emptied of liquid contents, and then filled with air or with gas while the colon is filled with water.

The deglutition murmurs may be auscultated just below the xyphoid cartilage while the patient swallows some water. The first murmur is heard at the very beginning of deglutition and is a hissing sound. The second murmur is audible six or more seconds later and is of a gurgling character. The first murmur is very often distinct, the second rarely so. Their presence is of no diagnostic value, but the absence of the second sound is to be noted in stenosis of the cardia.

The succussion splash may be sought for by shaking the patient while recumbent, or by striking over the region of the stomach with the finger-tips.

Various methods proposed for direct mensuration of the stomach are not very satisfactory. A stiff sound introduced should pass about twenty-four inches from the incisor teeth. The same objection, however, applies to this as to Leube's method of palpating the tip of the sound; that, namely, the conclusions are liable to be fallacious. It has also been recommended to fill the stomach with water and then to remove and measure the amount used; but it is difficult to determine just when the stomach is full. Other methods have been proposed, but have not found general acceptance.

Gastroscopy was recommended by Mikulicz, but it is impracticable for ordinary employment. A gastrodiaphane was invented and used by Einhorn. It consists of a small stomach-tube with a little electric light at its gastric extremity. The stomach should be distended with water and the tube introduced. The position and limits of the organ are denoted by the dull red light seen through the abdominal walls.

Chemical Examination of the Contents of the Stomach.—For this purpose it is necessary to remove the contents with a stomach-tube. This tube is preferably made of moderately soft rubber with a somewhat tapering extremity. It is open at the end, and has several large and small fenestræ near the tip. The tube should be thirty or more inches in length. In its introduction it should be pressed against the posterior pharyngeal wall guided, if necessary, by the fingers of the left hand placed in the mouth. The patient being told to swallow, the muscles of deglutition grasp the extremity of the tube, and, with a little forward pressure from the operator, it moves downward to the stomach. A slight resistance is felt as the tube passes the cricoid cartilage. If the pharynx be very irritable it may be painted with a solution of cocaine before the introduction is attempted. The tube should be introduced a distance of about twenty-four inches from the incisor teeth, except in cases of gastrectasia, where it is necessary to insert it somewhat farther. After introduction the contents of the stomach may be removed by expression, the patient leaning forward while firm pressure is made over the epigastrium, or the contents may be aspirated by means of a rubber bulb attached directly to the tube or mediately through a wide-mouthed, air-tight bottle, in which the gastric contents may be received.

Einhorn has invented a stomach-bucket for the same purpose, but only small quantities of the contents can be removed by it.

Different test-meals have been proposed, by the removal and examination of which the condition of the gastric chemistry could be determined. Among them are those of Riegel, Ewald, Reichmann, and Jaworski. The trial breakfast of Ewald is the one in common use. This consists of a roll of bread with a glass of water or a cup of tea without milk or sugar, taken on an empty stomach. In an hour the gastric contents are to be removed. These should normally not exceed 40 cc. in amount. Free HCl should be present, but lactic acid absent. Rennet ferment and rennet zymogen should be present. Starch should have been converted to acrodextrine, maltose, or dextrose, so that the iodine reaction for starch can no longer be obtained. Pepsin should be present; albumin should have been converted into albumoses.

The gastric contents should now be filtered and an examination of the filtrate conducted systematically. The filtrate should be clear yellow or yellowish-brown.

The *reaction* may be tested by litmus-paper or Congo paper, the latter becoming blue in the presence of acid.

The *total acidity* is a matter of importance. It is to be determined by titration. Ten cubic centimetres of the filtrate are titrated with a one-tenth normal sodium-hydrate solution, having previously added to the filtrate a few drops of a weak solution of phenolphthalein. The point of neutralization is indicated by the development of a persistent faint-red color. Ordinarily four to six cubic centimetres of the solution are required. If the acidity depends upon free HCl, and not on acid salts or other acids, the amount present may be calculated on a basis of one cubic centimetre of the one-tenth soda solution, equaling .03646 of HCl.

The determination of whether the acidity depends on *free acid* or on acid salts is done with tropæolin ōō, or with Congo red. The dark yellowish-red solution of tropæolin, or the paper dipped in it and dried, is turned to dark brown or brown-red with free acid. With acid salts, however, it becomes straw-yellow. Congo red is made deep blue with free acid, but is unchanged by acid salts.

To determine the *presence of HCl*, one of the best reagents is that of Günzburg. One part of vanillin and two of phloroglucin are dissolved in thirty of alcohol. The solution is pale yellow, and should be kept in a dark bottle. To employ it, one drop is placed in a porcelain dish with a drop of the gastric filtrate and the dish heated gently over the flame. If HCl be present, a rose tint develops as evaporation takes place, and finally becomes a deep cherry-red. The test is extremely delicate, and is not interfered with by albuminates, acid salts, or organic acids.

Another test is that of Boas, consisting of resorcinum resublimatum, 5; saccharum album, 3; spiritus dilutus, 100. Three to five drops are mixed with an equal amount of the filtrate in a porcelain dish and gently heated. A purple-red color is developed if free HCl be present.

The *actual amount of HCl present* may be determined by Boas's modification of Mintz's method. Ten cubic centimetres of the filtrate are shaken with one hundred cubic centimetres of ether to remove the organic acids. The fluid remaining after the ether is separated is titrated with a $\frac{1}{10}$ per cent. normal soda solution until Congo red fails to develop a blue color. In place of this the original method of Mintz may be employed, in which no ether is needed, and the neutralization is determined by the phloroglucinvanillin of

Günzburg. Ordinarily the amount of HCl varies between .14 and .24 per cent.

The best test for *lactic acid* is that of Uffelmann, consisting of a dilute aqueous solution of neutral ferric chloride, to which is added a drop or two of carbolic acid until an amethystine color results. A few drops of the gastric filtrate added to this changes the color to a canary-yellow if lactid acid be present. Lactates, phosphates, sugar, alcohol, and, if concentrated, HCl, may, however, produce the same yellow color. If, then, it be important to determine positively that the reaction is due to lactic acid, twenty cubic centimetres of the filtrate should be shaken with three or four different portions of ether, each of ten cubic centimetres; the ether evaporated to dryness; an aqueous solution of the residue made, and this tested by Uffelmann's method.

Butyric acid produces a more brownish-yellow color with Uffelmann's test. It has likewise a very characteristic odor.

Acetic acid also has a characteristic odor. It may likewise be detected by dissolving the residue of the ethereal extract in water, neutralizing this with carbonate of sodium, and then adding a neutral ferric-chloride solution, which produces with it a blood-red color.

If hydrochloric acid be present, it may be assumed that *pepsin* is also secreted. A further indication of it under these circumstances is trial of artificial digestion. A thin disk of hard-boiled egg-albumin is placed in a test-tube with a few cubic centimetres of the gastric filtrate, and kept at a temperature of about 100° F. for some time. If pepsin and HCl are present in normal amount, the egg will be dissolved in a few hours.

If no HCl be present in the gastric contents, *pepsinogen* will be found instead of pepsin. Enough of the acid should be added to produce the color reaction. This turns the pepsinogen, if present, into pepsin. The test of digestion may then be applied.

The *presence of rennet* may be determined by adding to a small quantity of neutral boiled milk an equal quantity of carefully neutralized gastric filtrate, and keeping this mixture at about 100° F. Curdling will take place in ten to fifteen minutes if the curdling ferment be present. In place of this method two to five drops of the filtrate may be added to ten cubic centimetres of raw milk and the same procedure followed.

The *rennet zymogen* will not itself curdle milk. Its presence may be detected by first rendering the filtrate alkaline with soda solution, and thus destroying any rennet present, and then by adding two or three ccm. of a 1 per cent. solution of chloride of lime. If the rennet zymogen was present, the filtrate will now coagulate milk if tested for rennet.

The tests for *peptone* and the various albumoses are too complicated for ordinary clinical study, and the subject is by no means clearly understood.

Starch and *erythrodextrine*, may be tested for by the addition to the filtrate of Lugol's solution. The first gives a blue color with iodine; the second, a purple.

Motor and Absorptive Power.—Finally, are to be considered the motor-power and the absorptive power of the stomach. The *motor-power* can be determined by noting the amount of fluid obtained after a test-breakfast. If more than forty cubic centimetres are procured, it is an indication that this power is diminished. Another method is that of the use of salol, recommended by Sievers and Ewald. Fifteen grains of salol are taken in water soon after a meal. In half an hour the urine is tested with neutral ferric chloride for the violet reaction which it produces with salicyluric acid. The testing may well be done on filter-paper. It should be continued every fifteen minutes until a result has been obtained. The color indicates that the salol has left the stomach and been broken up in the alkaline juices of the intestines into carbolic and salicylic acids. The reaction should appear in sixty to seventy-five minutes, and a longer delay indicates motor insufficiency. Huber modifies the test by determining how long the reaction persists. In healthy persons he claims that it should last twenty-four hours, while in cases of motor insufficiency it may continue two days. Neither method is entirely reliable, owing to variations in the composition of the contents of the first part of the small intestine.

The *rapidity of absorption* from the gastric mucous membrane may be determined, as recommended by Penzoldt, by giving fifteen grains of iodide of potash in capsules, and by testing the saliva every few minutes with starch and acid in search for the iodine reaction. The blue color should be produced within fifteen minutes in normal cases. The value of the test, however, is questionable.

ACUTE GASTRITIS.

Definition.—An acute inflammation of the glandular layer of the stomach.

SYNONYMS.—Simple gastritis; Acute gastritis; Acute gastric catarrh.

Etiology.—This exceedingly common affection, which includes not only severe gastritis but the mild cases of what is commonly called gastric catarrh, is due indirectly to a variety of causes. For instance, a distinct predisposition exists in certain persons, and this is sometimes clearly hereditary. Gout is also a predisposing cause. Numerous acute febrile diseases are accompanied by a simple acute gastritis. Nervous disturbances are not without influence through their alteration of the character of the gastric secretion and of the motor power of the stomach, and the consequent fermentation which is thereby permitted. Insufficient clothing or any other defect in hygiene acts as a cause in young children.

Ordinarily, however, the prime cause of acute gastritis is a direct local action upon the stomach of some irritant substance, usually the result of an error in diet. Sometimes a greater quantity of food is taken than the stomach at that time can digest, although the amount may not in itself be unusually large. This may produce a gastritis by mechanical irritation, the food being too long retained; or more probably by chemical irritation, the result of some product of decomposition. So, also, the quality of the ingesta may be impaired and produce disease. The excessive use of alcohol is a frequent cause. The use of very hot, or, particularly, very cold, substances, as ice-water or beer, sometimes acts in the same way. Articles of food or drink which are partially decomposed are particularly active in producing acute gastritis. This is especially true of milk given to infants during hot weather.

In the cases of most of the substances whose quality disagrees with the stomach the trouble is due to the action of certain microbes which have been swallowed with the food. Possibly in other instances the food possesses certain chemical ingredients, produced independently of any microbic action, which act as direct local irritants. When the irritation is due to the action of microbes producing fermentation, as it nearly always is, it is probable that the cause of the abnormal fermentation is a lack of perfect balance between the anti-fermentative gastric juice and the micro-organisms.

Pathology.—As was discovered by Beaumont, the mucous membrane of the stomach is red and swollen and marked with small hæmorrhages or even erosions. An increase of mucus is present. The submucous connective tissue is œdematous. Under the microscope is found a small-celled infiltration of the interstitial connective tissue, with swelling and granular change in all the glandular cells.

Symptomatology.—The disease is conveniently divided into *afebrile* and *febrile* gastritis. In the former the symptoms set in with a sensation of discomfort and flatulent distention of the abdomen, thirst, loss of appetite, belching, headache, depression, dizziness, nausea, and possibly vomiting. In infants this latter symptom is one of the most common. The tongue is heavily coated, and often indented by the teeth. There is frequently a bad taste in the mouth and the breath is offensive. Herpes labialis may be developed. The saliva is increased in quantity, the face is pale, the pulse more rapid than normal. There may be constipation, but later diarrhœa is frequent. The urine contains abundant urates. Vomiting is often a relief, and the symptoms may begin to improve after it. The vomited matter consists of the food not much altered, even though ejected several hours after ingestion. Considerable mucus is mingled with it. If vomiting continue, as is quite often the case, the ejected matter finally is composed of mucus and bile. The vomited material does not contain free HCl. Lactic and fatty acids are frequently present in it, but not always so.

The *febrile form* develops as does the other, but with symptoms much more severe and with fever from the beginning. The temperature may even reach 104° F. This condition has often been called "gastric fever," and Lebert has claimed that acute febrile gastritis is sometimes the result of a special infection.

Diagnosis.—The acute afebrile cases can be readily recognized. In the febrile form the diagnosis is not so plain. The gastritis may be the initial evidence of some other affection, such as one of the infectious fevers. The disease may also resemble meningitis, especially if headache and delirium be present. If the gastric pain be unusually severe, the disease may simulate bilious colic. It is, however, most liable to be confounded with the earlier stages of typhoid fever. In gastritis, however, the rise of temperature is sudden, the remissions less marked, and there are no rose-spots or enlargement of the spleen. Herpes does not occur, epistaxis is not common, and there is no bronchitis.

Prognosis and Course.—The affection lasts twenty-four hours to three to five days, and then ceases, unless it pass into the subacute or chronic form.

Treatment.—In the mildest cases treatment is scarcely needed, especially if vomiting has already taken place. If the attack be very severe and vomiting has not occurred, and if at the same time there are much nausea and gastric distress, with dulness on percussion over the stomach, indicating that the organ is still full, emesis may be induced by drinking warm salt water or by the ingestion of some mild emetic, especially ipecacuanha. Ordinarily, however, this is not needed. It is very important that the patient should do without food as far as possible for a day or two. The administration of bicarbonate of sodium with bismuth, and perhaps some aromatic, as oil of cloves or ginger, is of value for alleviating the persistent nausea and gaseous or sour eructations. The irritating gastric contents passing into the bowel are very liable to produce diarrhœa soon after the gastric symptoms have commenced. Should this not occur, or even if it have and it be thought that some of the material is still present, it is well to administer a laxative, as a seidlitz powder, calcined magnesia, or small divided doses of calomel, one-eighth of a grain every hour.

ACUTE PHLEGMONOUS GASTRITIS.

This rare affection, called also acute suppurative gastritis, may be either idiopathic or metastatic in origin; in the latter instance depending upon the exanthemata or puerperal infection or other septic condition. The cause of the idiopathic cases is not known, but it is clear that there has been an infection by pus-producing organisms. Men have been oftener attacked than women.

The pathological process may be either circumscribed or diffuse. In the former there is the production of an abscess which may attain the size of an egg, and which is situated in the submucous tissue, and perhaps infiltrates the adjacent layers to some extent. It may finally burst into the cavity of the peritoneum or that of the stomach. In the diffuse process the purulent infiltration spreads extensively into adjacent coats, causing fatty degeneration of the muscular layer and perhaps producing cribriform perforation of the mucous lining or of the serous coat.

The onset of the disease may be sudden or most insidious. The symptoms generally consist of violent burning pain in the epigastrium; dry tongue; thirst; loss of appetite; high fever of a septic type; rapid, weak pulse; vomiting, sometimes of pus; delirium; prostration; coma. Jaundice sometimes occurs; constipation may be absolute, but diarrhœa is more frequent.

The disease generally runs a violent course, although occasionally more chronic in character. It rarely lasts as long as two weeks.

The diagnosis can scarcely be made during life. Vomiting of pus might be produced as well by some suppuration external to the stomach perforating into its cavity.

Treatment can be only palliative. Cold may be applied to the abdomen, ice be swallowed, and stimulants and morphine be administered.

Acute Toxic Gastritis.

Definition.—A severe inflammation of the stomach the result of the ingestion of poisons.

Etiolgy.—The cause of the affection is the taking of such poisons as concentrated mineral acids, carbolic acid, oxalic acid, concentrated alkalies, alcohol, phosphorus, antimony, corrosive sublimate, cyanide of potash, chlorate of potash, arsenic, and the like.

Pathology.—These substances may act in different ways. Phosphorus, alcohol, antimony, and arsenic produce a fatty and mucoid degeneration of the glandular cells, with a small-celled infiltration of the entire glandular connective tissue which is very slow to disappear. The corrosive poisons, on the other hand, produce more or less gross destruction of the mucous membrane and sometimes of the submucous layer, and a black eschar may result. The muscular layer may exhibit a gelatinous or serous infiltration, or the whole thickness of the wall of the stomach may be involved and perforation take place. In the milder cases there are merely superficially necrosed areas here and there, with the surrounding mucous membrane hyperæmic and the submucous tissue hæmorrhagic and infiltrated with serum.

Symptomatology.—The symptoms in the case of non-caustic poisoning are variable, depending upon the kind of poison taken. In the case of the caustic poisons there is, besides the affection of the mouth, throat, and œsophagus, intense pain in the stomach, persistent vomiting of bloody mucus or pure blood, and sometimes of pieces of the mucous membrane. The abdomen is swollen and tender, the urine may contain albumin and even blood. Petechiæ sometimes develop. The pulse is weak, the skin cold and clammy; there are cyanosis, great restlessness, profound collapse, and sometimes convulsions.

Diagnosis.—The diagnosis depends generally upon the history of the case and upon symptoms other than those connected with the stomach. In the case of the caustic poisons the lining of the mouth and pharynx exhibits evidences of corrosion. The recognition of the special poison which has been taken is too large a subject for the present discussion, and may be better studied in works upon toxicology.

Prognosis and Course.—The prognosis depends largely upon the amount of poison ingested. If it be very small, recovery may follow in a few days. If a somewhat greater amount has been taken, the course is apt to be prolonged. Sloughs may separate and be vomited, and the ulcers which result may produce cicatricial contraction. There often remains severe, prolonged disturbance of the gastric functions. Death may occur during the course of these symptoms.

If the amount of the corrosive poison be large, death may take place in a few hours from collapse, or, in the event of the escape of pus, from perforation.

Treatment.—If the poison be not a caustic one, the stomach should be emptied as soon as possible unless vomiting has already been severe. Emetics may be given, but siphonage with the stomach-tube is to be preferred. In the case of the caustic poisons the stomach-tube should be avoided as a possible source of danger. If strong acids have been taken, the patient should be given calcined magnesia or chalk. If the poison be a strong alkali, vinegar is nearly always to be had as an antidote.

The treatment of the further course of the case is entirely symptomatic. In the worst cases only palliative treatment is indicated, including the administration of morphine.

MEMBRANOUS GASTRITIS.

This condition may occasionally be produced by diphtheria, scarlatina, variola, pyæmia, typhoid fever, and pneumonia. The pseudo-membrane may be either a necrotic one or it may be the result of a fibrinous inflammation. In diphtheria ulcers may be found which are usually situated at the cardia. The symptoms are masked by those of the primary disease. There is no means of determining the presence of the lesion during life.

PARASITIC GASTRITIS.

Apart from the invasion of the gastric mucous membrane by the bacillus of diphtheria and the pus micro-organisms of phlegmonous gastritis, both of which conditions might properly be described here, other parasites may occasionally be present and excite inflammation. A fatal case of the development of favus in the stomach is reported by Kundrat. A *bacillus gastricus* is described by Klebs. It had attacked the gastric glands, being found in their lumen and between the membrana propria and the epithelial cells. Anthrax has been found in the stomach, producing small areas of swelling and sloughing of the mucous and submucous layers. An emphysematous gastritis has been described by E. Fränkel, and attributed by him to the presence of bacilli. Tuberculosis and syphilis may occasionally involve the gastric mucous membrane. Sarcinæ and the yeast fungus are sometimes found in the stomach in cases of fermentation, and may possibly increase an inflammatory condition already present. Larger parasites may occasionally produce acute gastritis. Among those described are ascarides, tæniæ, earth-worms, cheese-maggots, and the larvæ of certain dipteræ.

CHRONIC GASTRITIS.

Definition.—A chronic inflammation of the mucous membrane of the stomach characterized by changes in the gastric juice, increased secretion of mucus, weakening of the muscular power, and the symptoms of chronic indigestion.

SYNONYMS.—Chronic dyspepsia; Chronic gastric catarrh; Atrophy of the stomach; Chronic glandular gastritis; Flatulent dyspepsia; Embarras gastrique.

Etiology.—The causes of chronic gastritis may be various. Repeated attacks of acute or subacute gastritis are liable eventually to terminate in the chronic disorder. The commonest cause of chronic gastritis is the persistent use of an improper diet. This is particularly true of fatty substances, especially if fried. Starches, too, if used in too large amount, are often difficult of digestion. Improper methods of eating are as deleterious as is improper food. Bolting the food with little mastication and the washing of it down with large draughts of ice-water are quite sufficient to set up a chronic gastritis. Whenever a number of the teeth have been lost or are badly decayed, so that efficient mastication is impossible, there is great danger that chronic gastritis will be induced. The use of alcohol and, to some extent, of tea and coffee and tobacco, is a frequent factor.

Scarcely less important than dietetic errors, rank the neglects of hygiene which favor repeated congestions of the mucous membranes, by imprudent exposures or by sudden checking of the action of the skin. Insufficient clothing, exposures to damp, and draughts, ill-drained and damp residences are thus among the frequent causes of chronic gastritis.

Another important series of predisposing causes is that including various constitutional disturbances; among them being Bright's disease, diabetes, tuberculosis, gout, uterine disease, and anæmia of any form.

Chronic gastritis may result from interference with the circulation of the blood in the stomach, producing passive congestion of the organ. This may occur in heart disease and in cirrhosis of the liver. The disease may also attend such local lesions as ulcer, cancer, and gastrectasia.

Pathology.—In the cases of moderate severity the mucous membrane, particularly toward the pylorus, is of a pale gray color, with scattered areas of injection, patches of ecchymosis, and evidences of pigmentation. It is thickened and is covered with a whitish, tenacious, firmly adherent mucus. The swelling of the mucous membrane causes it to be elevated here and there in places, and produces a warty and papillary condition to which has been applied the name of *surface mamelonée.* When the change is excessive, it may even produce polypoid outgrowths which may reach the size of a pea and which project upon broad or narrow peduncles. The pigmentation and the mamillary condition are best marked near the pylorus. Small erosions and follicular ulcers of the mucous membranes are frequently met with. The submucous and muscular layers may also be thickened, particularly toward the pylorus, and may even produce stenosis here.

The microscopical changes consist in alteration both of the connective tissue and of the glands. The cells of the latter are enlarged, show cloudy swelling, or are in part atrophied. The principal and parietal cells cannot be distinguished from each other. The tubes are dilated, are of irregular form, and exhibit numerous branching diverticula. Cysts may be seen close to the submucosa, resulting from the cutting off and occlusion of isolated glands. They may be empty or filled with remnants of hyaline epithelium. The epithelial cells, even far into the fundus of the peptic glands, show various stages of mucoid degeneration.

Besides these degenerative changes there is a small-celled infiltration, most abundant near the surface of the mucous membrane, which lies between the glands and presses them from each other. There is also an increase in the growth of connective tissue, which sends up prolongations from the submucosa between the glands.

From this condition the process may advance to one of two forms of atrophy of the mucous membrane of the stomach. In the first, which may be called *simple atrophy*, or *phthisis ventriculi*, there is a progressive fatty degeneration of the glandular cells with a steady increase in the interglandular connective tissue. In well-advanced cases the mucous membrane is entirely destroyed, and nothing remains but a thin layer of small round cells and fibrous tissue. Cysts can be found here and there in the destroyed mucosa. The mucous membrane appears to the naked eye as a smooth, thin, white surface. The stomach is of normal size or larger than usual, and its walls are thin.

In the second form, the *cirrhotic atrophy*, the glandular destruction is the same, but there is in addition a tremendous increase in the thickness of both submucous and muscular layer, the result of overgrowth of both connective tissue and muscular fibres. The whole organ diminishes in size very greatly, and may be able to contain no more than a few ounces.

Symptomatology.—According to the classification of Ewald, three clinical forms of the disease can be readily recognized: 1st. *Chronic simple gastritis;* 2nd. *Chronic mucous gastritis;* 3d. *Atrophy of the gastric mucous membrane.* The last is but a final stage of either of the other two. The recognition of the other two forms depends largely upon an examination of the gastric contents, and can best be considered under Diagnosis.

The earlier subjective symptoms are much the same in the two varieties. The chief complaint of the patient is of the persistence or frequent recurrence of evidences of indigestion. There is an unpleasant taste in the mouth. The tongue is generally coated, either uniformly or with the exception of the edges and tip, which are redder than normal. Its edges are frequently tooth-marked. Aphthæ are sometimes present in the mouth. The secretion of saliva is often increased. The appetite is variable; sometimes good, often impaired. Thirst is usually increased, especially while food is being ingested. Eructation of gas is very common, and takes place as a rule after eating. It is sometimes but temporary and inconsiderable, but often lasts for hours and is a source of great annoyance. The gas may be odorless or may be offensive. Eructation of fluid or of solid portions of food may accompany belching. The regurgitation of clear watery fluid is called water-brash, or pyrosis. There is often distention of the abdomen after eating, with a sensation of oppression and distress which may develop into actual dyspnœa. The region of the stomach is diffusely and not greatly tender to pressure. In children who have passed the first dentition persistent abdominal distention is a very common symptom of chronic gastritis.

Actual pain in the stomach may be present, but is not common. There may, however, be colicky pain in the intestines. When the pain is localized beneath the lower portion of the sternum or in the region of the heart, it is often called heart-burn or cardialgia. It may depend upon irritation of the cardia and œsophagus by fluids which have been regurgitated.

Nausea is not uncommon, and vomiting may occur at very irregular intervals, although generally at some little time after meals or else in the morning before breakfast. Many patients rarely suffer from it, although other symptoms of the disease are present in full force. The vomited matter varies in character according to the form of the disease present. Hydrochloric acid is usually absent from it or diminished in amount, and mucus and abnormal acids may be present, together with partially digested food.

The diminished secretion of hydrochloric acid not only produces a delay in digestion and a consequent prolonged retention of food, but allows abnormal decomposition to take place which distends the stomach with gas. The decomposing gastric contents act as an irritant, and cause a contraction of the sphincters. This still further increases the retention of food, while the gas produced presses upon the gastric walls and paralyzes them. In this way a condition of atony of the muscular fibre of the organ is brought about, and even dilatation may ensue. The absorptive power of the stomach is retarded, and iodide of potash given will not appear in the saliva for perhaps over half an hour, instead of in fifteen minutes, as is normally the case.

Constipation is generally present in chronic gastritis, although some cases may exhibit diarrhœa at times. The urine is diminished in amount, high colored, and contains an excess of urates. The disposition is often altered, and the patients become irritable, depressed, and even melancholic. They are indisposed to exertion both of mind and of body, are inclined to sleep, and often suffer from headache. A hacking cough is often present, and has been called "stomach cough." It has probably no direct connection with the stomach itself, but depends upon an attendant irritation of the pharynx, possibly the result of regurgitation of acid matters. There is not infrequently some elevation of temperature; palpitation of the heart may be present; the pulse is small and sometimes retarded. Vertigo is quite common, especially with sudden movement, such as leaning over or rapidly straightening up again. It is generally slight, but sometimes severe enough to be a cause of great annoyance.

The final stage of the two forms of chronic gastritis described is atrophy of the gastric mucous membrane, or *anadenia*. It presents symptoms somewhat characteristic, although far from uniform or typical. It is oftenest seen in those who have reached or passed middle life, but may occur in the young also. If the process be at all extended, there develops an entire lack of digestive power of the stomach. The symptoms are those of very severe dyspepsia together with the evidence of decided failure of nutrition. There is great distress after eating. Vomiting is often a very prominent symptom, and there is progressive and great loss of strength and often of flesh, so that the case indicates the existence of cancer. In other instances the patients do not lose much flesh, but become so extremely anæmic that the complex of symptoms is almost or quite identical with that of pernicious anæmia—a fact to which attention was first called by Flint. In the cases of atrophy attended by cirrhosis these same symptoms may be present. There is also exhibited an inability to take more than a very small amount of nourishment at one time.

What might almost be called a separate form of chronic gastritis is the disease as seen in infants. In these cases there is vomiting, at first of all food as soon as taken, but occurring later even between the periods of feeding. The ejected matter is transparent and watery, and even sudden movement of the child will induce vomiting. The abdomen is distended, there is much abdominal pain, the bowels are constipated, the child rapidly wastes, the face becomes pinched, and cerebral symptoms finally develop.

Diagnosis.—The diagnosis of chronic gastritis can only be made by excluding the existence of such affections as cancer, dilatation, ulcer, and the various neuroses of the stomach. This exclusion is often a matter of great difficulty, inasmuch as the symptoms of these various diseases are by no means always characteristic. Thus, a patient who appears to be suffering from gastritis may begin to improve only when the treatment directed to a neurosis of the stomach is instituted. To make a diagnosis it is sometimes necessary to use all means at our command, including careful examination of the gastric secretions. This chemical examination is likewise the method to be employed to separate the forms of chronic gastritis from each other.

In *chronic simple gastritis* the stomach, while fasting, contains a small amount of thin mucous fluid of yellowish color. The contents removed after a trial breakfast in the manner already described show an acidity which is normal or diminished, a diminution of HCl, and often the presence of lactic acid and of fatty acids. Pepsin and rennet are lessened in quantity and propeptone and peptone are present.

In *chronic mucous gastritis* the chief diagnostic feature is the presence of a large amount of mucus in the gastric contents after the trial breakfast. HCl is generally absent; the acidity is diminished; peptone is in minute amount but propeptone abundant; pepsin and rennet are each much diminished. A large quantity of mucus is present in the stomach even when fasting.

In *atrophy of the gastric mucous membrane* the gastric contents after the trial breakfast contain no mucus, HCl, pepsin, or rennet. When the patient is fasting the stomach is generally entirely empty.

The various forms are not always sharply separated, and transition stages between them exist. It must also be remembered that absence of HCl may also occur in cancer and in some of the gastric neuroses, so that even with the chemical examination the diagnosis of chronic gastritis is not always easy.

In infants chronic gastritis is to be distinguished from tubercular meningitis. The sunken fontanelle and absence of fever will aid in doing this.

Prognosis and Course.—The course of chronic gastritis is remarkably slow. The disease improves or even disappears for a time, and then suffers exacerbations or relapses upon very slight cause. A slight indiscretion in diet or slight impairment of the general health of whatever nature, may be all that is necessary to bring back the symptoms in full force. The prognosis as regards complete recovery must therefore be guarded if the disease has lasted any great length of time. As regards duration of life, it is for the most part favorable, although not entirely so. In severe and long-continued cases there is always a possibility of atrophy developing. This condition in greater or less degree is much more frequent than is ordinarily supposed, and may be the cause of many of the deaths attributed to old age. There is also the possibility of decided and permanent impairment of general nutrition by chronic gastritis, even without the condition of atrophy having been reached.

Treatment.—The treatment of chronic gastritis may be divided into (1) Dietetic and hygienic; (2) Medicinal.

(1) *Dietetic and hygienic treatment.*—It has been truly claimed that every dyspeptic must be his own physician. The cure certainly lies largely in the determination to follow exactly the dietetic and hygienic regimen which may be prescribed. Regularity in the time of meals is of the greatest importance, as is also thorough mastication of the food. The freedom from worry or from mental effort during the time of meals or immediately afterward is conducive to proper digestion. It is a common observation that dyspeptics can at times partake with impunity of comparatively indigestible dishes, but that at other times, when business or other care or any impairment of the general health is present, much simpler food is productive of troublesome gastric symptoms.

As a general rule, no hearty meal should be followed at once by mental or physical exertion. The time of day at which the heartiest meal (dinner) should be taken should be determined largely with this truth in mind. Nevertheless, the element of idiosyncrasy is very powerful in this connection. So, too, there exist remarkable idiosyncrasies toward certain articles of food, in that certain patients can digest what would be deemed entirely unsuitable for them, and, on the other hand, may not be able to bear articles of diet generally of the blandest and most unirritating character.

In mild cases of chronic gastritis, therefore, the experience of the patient is to be duly considered and too rigid a regimen is to be avoided. It is difficult or impossible to outline any general rules, since cases vary so greatly in their requirements in this regard. By close observation on the part of the patient those articles of food which disagree should be noted and banished from the dietary. As a rule, fat in any form, highly seasoned and very sweet dishes, pastry and all hot bread should be avoided altogether. When there is difficulty in digesting albuminoids, it is important to choose those which are most easily assimilated. Tendinous or tough meat, hard-boiled eggs, pork, young veal and flesh of animals which have just been killed should all be avoided. The use of fish must depend upon the idiosyncrasy of the patient. Sometimes smoked or salt meats are better borne than fresh articles. Starchy substances are often particularly hard to digest on account of the abnormal fermentation which they undergo in many instances of chronic gastritis. In cases, therefore, characterized by much flatulent and acid eructation starch should be avoided. This is especially true of potatoes, which very many patients find it entirely impossible to eat with comfort. On the other hand, starch which has been changed to dextrin or which has been thoroughly baked and dried after baking is frequently well borne. It is on this account that dry toast and zwieback often constitute such valuable articles of diet.

The question of the employment of fruits and green vegetables admits of different answers, according to the individual case. Cabbage and cauliflower are seldom, if ever, to be taken. Beans and peas, if ripe, are often productive of fermentation, but if young and well cooked are usually well borne. Most ripe fruits can generally be eaten, particularly if cooked. Whatever the kind of food ingested, care must be taken that the patient stop short of the entire satisfaction of hunger, and allow rather long intervals between meals in order that the stomach may have sufficient time to empty itself.

In severe cases an absolute milk diet may be given for a time. It is generally easily digested, though patients are sometimes met with who cannot take it in any form. It may be given raw, boiled, with lime-water, in the form of buttermilk, or peptonized. From one and a half to two and a half quarts are needed in twenty-four hours. It is often impossible to administer enough nourishment in this way, and the diet must be supplemented to some extent. It should be remembered also, that during the continuance of the diet, the patient must restrict considerably his usual exercise. Very frequently some of the various peptonized meat products may be given with advantage. This is especially true in the very severe cases in which the power of digestion seems almost entirely wanting. In atrophy of the gastric mucous membrane predigested foods often serve a useful purpose. Fluids with meals should be taken in only moderate quantity in order not to dilute the gastric juice too greatly. They should be neither very hot nor very cold. Tea and coffee are best omitted, and concentrated alcoholic beverages are harmful. Light wines, especially if dry, are often beneficial, but should not hastily be recommended for constant use.

Conjoined with a proper diet, attention must be given to general hygiene. An abundance of fresh air and of exercise is essential. At the same time constant care is required to avoid renewal of gastric irritation from atmospheric influences. The system is unduly sensitive in such cases and the mucous membrane of the stomach is the most vulnerable point. The tone of the skin should be improved by cold douches or cool sponge baths followed by brisk friction. Massage may also be useful. The dress should be carefully regulated. Exposure to damp and draughts should be avoided. Means must be taken to overcome the despondency so frequently present. Change of air, travel, sea-bathing, etc. will often accomplish more than almost any other measures employed. Prolonged sojourn at the various mineral springs is of distinct benefit, both on account of the change of air and scene, and on account of the careful diet which, in some places at least, is prescribed and enforced. The action of the water is of least importance, although a decided factor in suitable cases.

(2) *Medicinal treatment.*—This may be classified into measures intended (*a*) to supply the deficient chemical elements of the digestive secretions; (*b*) to restore the secretory and motor power; (*c*) to prevent fermentation and control special symptoms.

(*a*) Since in chronic gastritis hydrochloric acid is diminished or wanting, the natural indication is to supply this want by the administration of an acid. By no means all cases, however, are benefited in this way, and proper discretion must be employed, since a predominance of certain symptoms may demand other treatment. The dilute hydrochloric acid should be given in doses of 6 to 15 drops, further diluted with water, shortly after eating. It is often given advantageously in combination with small doses of quinine and strychnine as follows:

℞. Quiniæ sulph.,	gr. xxx;
Strychninæ sulph.,	gr. ½;
Acid. muriatic dilut.,	fʒijss;
Tr. cardamomi comp.,	fʒijss;
Aquæ,	q. s. ad f℥iv.

Dissolve and filter.

Sig. A teaspoonful in water after meals.

Not only does it aid in the digestion of food by its own action, but it converts pepsinogen, which may be present, into pepsin, and thus gives it an opportunity to exert its power. It is also an antiseptic and prevents fermentation. It is especially indicated where there is evidently a difficulty in the digestion of albuminoids. It seems questionable, however, whether it can be of any value in cases of complete gastric atrophy, since it is then unable to stimulate the secretion of pepsin, without which digestion cannot take place.

Pepsin has been very largely given in chronic gastritis. It is doubtful whether it is often indicated, since the hydrochloric acid administered is often all that is needed. Although hydrochloric acid is frequently absent, pepsin is generally present, except in mucous gastritis and atrophy of the gastric mucous membrane. Should an examination of the gastric contents show an absence of digestive power even after sufficient acidulation, pepsin should be given with acid three times a day after meals.

In some cases pancreatin may be administered in 5-grain doses alone or combined with bicarbonate of sodium. It is indicated where it is desired both to digest the albuminoid and the farinaceous element of the diet. It is also to be selected in cases of distinct atrophy, since in this condition no secretion of pepsin or of acid can be expected. Diastase in the form of malt extracts of syrupy consistence is very serviceable.

(*b*) To increase the glandular and motor power of the stomach, the cause of the gastritis is to be removed when possible. Such constitutional disturbances as Bright's disease, venous stasis from heart disease, anæmia, and malnutrition of any sort demand treatment.

The best means of direct treatment for the insufficient glandular activity is the employment of lavage. This is especially true of the mucous variety, since the great abundance of mucus in this form of gastritis coats the lining of the stomach and the food ingested, and thus interferes with the production of gastric juice, neutralizes it when formed, and prevents it from coming into contact with what has been eaten. It is not to be understood that it is desirable to resort to lavage promptly in all cases. On the contrary, there are many instances in which, after the institution of a strict diet and hygienic regimen, the symptoms yield promptly to suitable remedies without recourse having been had to the more radical measure of lavage. But where in spite of such treatment the gastritis proves obstinate systematic lavage may be associated with advantage. The washing should be done once every day or every other day, and preferably in the morning while the patient is fasting. If there be much mucus, a weak solution of bicarbonate of sodium (3 per cent.) or of chloride of sodium (1 per cent.) may be substituted. For excessive fermentation the washing with simple water may be followed by lavage with an antiseptic solution, as of boric acid or diluted carbolic acid. Instances have been reported, however, in which toxic symptoms ensued from the use of antiseptic solutions, particularly when allowed to remain in the stomach.

Where the symptoms are not severe enough to demand lavage, or where the patient objects to the treatment, much good may be accomplished by drinking one or two glasses of hot water a half hour or more before breakfast, or possibly before each meal. Alkaline water is even more serviceable, as it loosens the mucus more effectually.

The application of electricity, has been recommended, but is of questionable value unless applied internally in the form of faradism by means of an Einhorn electrode. Used in this way it may improve the tone of the muscular fibre and stimulate secretion. It is obvious, however, that the treatment is so unpleasant and irksome as to be applicable only to extreme cases. The stomach, previously emptied, should be filled with water and the electrode then swallowed. The electrode is attached to a very small rubber tube through which a wire runs connecting the instrument with the battery. The flat external electrode should be applied to the epigastrium, and only such a strength of current should be employed as can be felt slightly. Gentle traction on the rubber tube removes the instrument when desired.

Among the medicines employed to stimulate secretion the bitter tonics have long held an important place. Various experiments made with them have led to contradictory results, some observers maintaining that they are worthless, and others claiming that they have a distinctly positive action. Clinical experience has shown conclusively their value in properly selected cases. Nux vomica is probably the most useful, though quassia, gentian, ipecacuanha, and condurango are also of service. They are often best given some time before meals, but may be combined with hydrochloric acid and given after eating, as in the formula given above.

Nux vomica has an action apart from that of a stomachic in that it especially increases the muscular tone of the stomach.

Inasmuch as the production of hydrochloric acid depends upon the ingestion of chloride of sodium, it is often well to increase the amount of this taken in cases of chronic gastritis, since the acid is diminished in this condition. Nitrate of silver has been widely employed. It is one of the most reliable remedies in this form of gastritis. The dose and mode of administration must vary with the special indications of each case. It may be given in solution in distilled water on an empty stomach a half-hour before meals in the dose of gr. $\frac{1}{12}$ to gr. $\frac{1}{4}$: or in pill form, either before or after meals, and alone or combined with suitable doses of belladonna, nux vomica, or ipecac. Arsenic is frequently of value, and in the chronic gastritis of infants is one of the most serviceable remedies at our command. Orexin, recommended by Penzoldt, has not proved of the value which was anticipated for it. The administration of bicarbonate of sodium or some other alkali, or of an alkaline water a half hour before the meal stimulates the secretion of the gastric juice. This is, of course, at first neutralized by the alkali, but that which comes later is greater in amount than would otherwise have been produced. Especially to be recommended are the saline and alkaline (non-laxative) mineral waters.

Very many cases are so annoyed by fermentation and consequent flatulence that treatment must be primarily directed against this condition. If regulation of the diet be not of itself sufficient to correct the trouble, and if lavage cannot be employed, relief can be obtained by the administration of such aromatic and antiseptic drugs as creasote, thymol, the essential oils and oleoresins, salol, hydronaphthol, bismuth, and naphthalin. Bicarbonate of sodium may be advantageously combined with an aromatic, and the addition of a few drops of chloroform adds to the efficiency. Such a combination will very successfully relieve abdominal pain and distention, and prevent acid and gaseous eructations. Some such combination as the following is often of value:

℞. Creasoti (pure beechwood).,		gtt. xv;
Sodii bicarb.,		gr. clx;
Pulv. acaciæ,		
Sacchari,	*āā*. q. s.;	
Sp. Lavandulæ comp.,		fʒij;
Aquæ,	q. s. ad.	f℥iv.—M.

Sig. A teaspoonful in water after meals.

Although bicarbonate of sodium given in this manner after meals is theoretically improper, the clinical results are frequently most satisfactory, both temporarily and permanently. Nevertheless, it would be better to depend in most cases upon hydrochloric acid for an antifermentative action.

Vomiting may be treated by bismuth alone or combined with aromatic powder. Creasote, nitrate of silver, salicylic acid, carbolic acid, cocaine, hydrocyanic acid, and chloroform are also useful. Vomiting is not often a troublesome symptom, except in the chronic gastritis of infants.

Constipation, so frequently present in chronic gastritis, must receive efficient treatment. As far as possible this condition should be remedied by hygienic measures. Systematic muscular exercise and diaphragmatic respiration; sponging with cool water; brisk friction and massage; and careful regulation of the diet will often suffice to restore gradually the regular activity of the bowel. Many dyspeptics acquire a morbid anxiety as to the state of their bowels which should be dispelled by encouraging advice. It is by no means necessary in all cases that a daily evacuation should occur. Laxatives should as a rule be avoided as far as possible. A simple enema on alternate days may suffice until with the relief of the gastric trouble a more vigorous tone is acquired and renders any such assistance needless, a glass of hot water sipped before breakfast may prove adequate; or its action may be aided by the addition of a teaspoonful of common salt or of Carlsbad salt. These simple draughts like other mild saline mineral waters not only loosen the bowels, but at the same time act mechanically by washing out the stomach. The employment of such laxative waters, containing sulphate of sodium, as most of them do, must not be too long persisted in, nor must too large quantities be taken, since there exists the possibility, especially in anæmic and nervous subjects, of increasing the symptoms and depressing the strength. If, however, the gastric condition be secondary to disease of the intestine or liver, much good may be expected from the laxative salines.

NEUROSES OF THE STOMACH.

Nervous Dyspepsia.

Definition.—Distressed sensations after eating, with or without disturbance of the act of digestion, depending upon functional disorder of the stomach.

Synonyms.—Gastric neurasthenia; Atonic dyspepsia.

Etiology and Pathology.—As already stated, this condition is so allied to forms of gastritis in many of its symptoms that it is almost impossible to distinguish between them. There exist very numerous cases in which the nervous element largely dominates, although gastritis of mild degree is actually present. These cases are best placed in the category of nervous dyspepsia. On the other hand, the disease under consideration is itself a complex and varying condition, since it depends upon various more elemental gastric neuroses, not all of which are present in any one case. Thus there may be a hypersecretion of gastric juice or a hyperacidity merely; or there may be diminution of its secretion, or, again, more or less loss of motor power, or, finally, some sensory disturbance of the organ. It is consequently impossible to give any one clinical picture which will illustrate all cases of nervous dyspepsia.

Some general neurasthenic manifestations usually precede or accompany the gastric symptoms, indicating that the cause depends largely upon a neurotic constitution or upon nervous prostration. Mental or bodily exhaustion of any sort may produce the disease. Among these causes must be mentioned prolonged business or household cares and anxieties, sexual excesses, hysteria, and the neurasthenic state in general. Such diseases as tabes dorsalis and affections of the brain may be accompanied by functional disturbance of the stomach. The digestive disorders attending chlorosis and anæmia are often of the class of nervous dyspepsia.

According to Leube, three forms of nervous dyspepsia are to be distinguished: (*a*) that in which the secretion of the stomach is normal in quality and quantity; (*b*) that accompanied by diminution of acidity; (*c*) that with hyperacidity. The difference in pathological condition in these three forms depends upon the difference in secretion, which can only be accurately determined by chemical examination. The form with hypersecretion or hyperacidity of the gastric juice is a comparatively common one. Hyperacidity may depend upon hysteria or be a reflex disturbance from the presence of renal calculi or gall-stones. The condition may also, though rarely, be a paroxysmal one, and may then be entirely independent of food. It may be the result of great mental excitement, emotional disturbance, extreme neurasthenia, or locomotor ataxia. The relation of hyperacidity to gastric ulcer will be discussed later.

Nervous dyspepsia associated with diminished secretion may attend neurasthenia, hysteria, and sometimes locomotor ataxia. Diminution of acidity may also be observed, as already stated, in cases of chronic gastritis. Its occurrence in cancer of the stomach will be referred to again.

Besides the changes in secretion mentioned no pathological anatomical alterations are present in nervous dyspepsia.

Symptomatology.—The symptoms vary to a considerable extent. There is almost always discoverable some other evidence of a general neurotic condition. As regards the stomach itself, there are distress and uneasiness, particularly after eating, and yet examination with the tube seven hours after a meal shows the stomach to be empty, digestion having been completely accomplished, as it should normally be by this time. This test is, however, not to be entirely depended upon. The patient may be emaciated and anæmic or may be well nourished and apparently robust. Intestinal symptoms are very frequently associated with the gastric disturbance, since the general neurasthenic condition is liable to involve both portions of the tract. Constipation is common; flatulent distention of the intestine is the rule, and there may be some abdominal pain. Malaise, vertigo, headache, anorexia, sleeplessness, weakness, and mental depression may be present. In the form of nervous dyspepsia, which is combined with hyperacidity, there is a burning, gnawing pain in the stomach, thirst, and sometimes sour eructations, and even vomiting of acid liquid occurring while the patient is fasting, as, for instance, during the night or early in the morning. In the form with diminution of acidity there may be, according to Leube, pronounced symptoms of nervous dyspepsia, and yet the stomach will be entirely empty of food seven hours after ingestion.

Diagnosis.—The diagnosis of nervous dyspepsia from other forms is not always easy. The fact that the stomach is empty seven hours after eating is a strong argument in favor of its existence, although it is far from conclusive for every case, since the presence of food in the stomach depends not only upon the degree of digestion which has taken place, but also upon the existence of atony of the gastric walls. The relative amount of hydrochloric acid present is of no value from a diagnostic point of view, except as regards the form of nervous dyspepsia with which we may have to do. There is not so much pain as in gastric ulcer or cancer, and it is more diffuse in character. There is also not the very great localized tenderness present in ulcer. Vomiting, too, is not common in nervous dyspepsia.

But, after all, the conclusion that the dyspepsia is of nervous origin must usually be based upon the general history of the case and the aggregation of symptoms which point in this direction.

Prognosis and Course.—The course and prognosis of nervous dyspepsia is very uncertain. Some cases last a very short time and yield readily to treatment; others at last recover, and still others are absolutely resistant. The severity of the symptoms is not at all proportionate to the obstinacy to treatment.

Treatment.—The treatment of nervous dyspepsia must be directed partly to the stomach itself, and very largely to the general condition of the patient. The use of tonic measures, such as change of air and the administration of iron, quinine, strychnine, or arsenic, each in suitable cases, is nearly always indicated. When there is decided general neurasthenia or hysteria, a course of rest-treatment may prove most effectual. Such drugs as hydrocyanic acid, bromide of potash, cyanide of potash, valerian, and nitrate of silver, alone or in suitable combination, will often render great service. Thus, for instance, there may be given after meals one of the following pills:

℞. Argenti nitratis,	gr. iij;
Ext. belladonnæ,	gr. ij;
Pulv. ipecac.,	gr. xij;
Ext. glycyrrhiza,	q. s.

Ft. mass et div. in pil. xxvj.
Sig. Use as directed;

and two hours after meals from one-half to one teaspoonful of the following in water:

℞. Acid. hydrocyanic. diluti,	fʒj;
Elix. valerianatis ammoniæ,	f℥iij.—M.

Sig. As directed.

When there is decided increase of HCl in the gastric secretion, it is often necessary to administer an alkali to relieve the heartburn and the acid eructation. As the excess of hydrochloric acid interferes with the digestion of starch, patients with this form of nervous dyspepsia are sometimes benefited by using largely albuminoid diet. Steaks made of minced beef, barely cooked, are well suited for this purpose.

It is very important to relieve the constipation which so frequently attends the disease, but it is not advisable to administer purgative medicines. The remarks already made are applicable here also. The cultivation of a regular habit of evacuating the bowels and the employment of food of a slightly laxative nature are often efficient, and, when not, small doses of cascara will find a fitting place.

GASTRALGIA.

Definition.—A sudden, severe paroxysmal pain located in the epigastrium, and caused by irritation of the sensory filaments of the gastric nerves.

SYNONYM.—Gastrodynia.

Etiology.—The numerous different causes of this sensory gastric neurosis may be classified in several groups. In all cases the pain is the result of a direct or reflex irritation of the gastric filaments of the pneumogastric nerve. In the largest class of cases the disease is a functional neurosis, and depends upon some general condition, as neurasthenia, anæmia, certain of the psychoses, and disordered menstruation, especially at the menopause. This form of gastralgia is consequently oftenest seen in women, since they are the most frequent subjects of the general disorders mentioned. It may, however, occur in men in apparently perfect health in other respects.

Another class of cases depends upon some distant lesion of the nervous system to which the affection of the gastric nerves bears only the relation of a secondary reflex disorder. By far the commonest cause of this nature is locomotor ataxia, which produces the well-known gastric crises through involvement of the pneumogastric centre or trunk. Nevertheless, these crises may occasionally be brought about by other diseases which involve the same region.

Still another series of cases depends upon local causes. Here is to be placed the gastralgia resulting from hypersecretion of hydrochloric acid; for, although the prime factor is, of course, a neurosis, the actual cause of the pain is the local irritation of the terminal filaments of the pneumogastric nerves in the stomach by the acid. Peptic ulcer and cancer may produce gastralgia, as may less severe lesions of the gastric mucous membrane.

The excessive use of tobacco is a fruitful cause of gastralgia. Its action is partly local, especially when the objectionable habit of chewing tobacco is practised, and partly indirect through the effect of the drug on the nerves. The excessive use of coffee and tea may induce gastralgia in the same way.

Symptomatology.—The symptoms of all cases are tolerably uniform and quite characteristic. The attack is paroxysmal, and not infrequently more or less periodical. This is especially true in gastric ulcer, in which pain is sometimes liable to develop at night. It is nearly always independent of the ingestion of food, though not invariably so. Salivation or a sensation of discomfort may usher it in, or it may commence very suddenly. In well-marked cases there quickly develops an intense cutting or boring pain which may be limited to the epigastrium or may be more diffuse, passing around the lower ribs in the intercostal spaces toward the back. Sometimes it simulates a girdle sensation, while in other instances it extends upward over the sternal region and may be attended by some pain or aching in one or both arms. Vomiting is not common, excepting in the gastralgia of tabes, where it is liable to occur. Ingestion of food often relieves the pain. Moderate pressure on the epigastrium may give relief, while deep pressure is sometimes painful. The attack may disappear slowly or suddenly, and may leave the patient exhausted or at least relaxed. Often there is experienced a decided sensation of hunger after it. The attacks may be severe or mild, may last for hours or a very much shorter time, and may occur regularly at a fixed interval after each meal, or more usually only once a day, or even at intervals of several days or several weeks.

Diagnosis.—The diagnosis is often a matter of much difficulty, since the causes of the affection are so varied. The existence of cancer or of ulcer must be sought for, and the possibility of the disease depending upon locomotor ataxia must also be entertained. In all such cases the existence of other symptoms besides gastralgia will aid in the diagnosis. In cases dependent upon functional nervous disturbance of the stomach the slight connection which the attack has with eating or the entire absence of this association is a very notable feature. Sometimes, however, the pain is relieved by eating, and this, too, points to gastralgia, although not absolute proof of it. The existence of the tobacco habit in a marked degree is a suggestive fact. The association of the attacks of pain with symptoms of neurasthenia, hysteria, and the like indicates their gastralgic nature.

Prognosis.—This depends chiefly upon the cause and the possibility of its cure. With its removal the gastralgia disappears. When not associated with any organic condition the prognosis is usually good.

Treatment.—The treatment must begin with the detection, and, if possible, the removal, of the cause. Faulty habits of eating must be corrected. If any articles capable of producing the disease are used in excess, such as coffee, tea, tobacco, alcohol, candy, they must be prohibited. It is quite useless to begin the treatment without insistence on this point. It is often desirable to lessen somewhat the amount of food taken at each meal, and to secure its more thorough mastication, and then to add a certain amount of milk, say from four to six ounces, with a tablespoonful of lime-water, to be taken two hours after meals in advance of the time when the pain is to be feared. The general condition of the patient requires careful study, and much the same treatment is required as has been described under Nervous Dyspepsia. Arsenic is of peculiar value, and when the mucous membrane of the stomach is not irritable, it may be given in ascending doses, and often acts almost in a specific manner. If it is not well borne, nitrate of silver or valerianate of zinc, with small doses of opium and belladonna, is apt to be effective. In either case bismuth is usually desirable in full doses two hours after meals. There is a strong temptation to resort to opium or morphine for the relief of pain; but as the opium habit is readily formed in such cases, its use should be carefully guarded. Cocaine is sometimes of service, and Hoffman's anodyne has long been used to relieve gastralgic pain. Hydrocyanic acid, alone or with valerin, may avert the attacks and gradually improve the condition of the affected nerves. Chloroform in doses of from 5 to 10 drops is frequently useful. In case of a severe attack, the more prompt of these remedies must be tried, and, if relief be not obtained, chlorodyne or morphine must be administered. Ice may be swallowed or cold compresses or mustard plasters be placed over the epigastrium.

The local application of the galvanic current, uninterrupted or slowly interrupted, has proved valuable even in obstinate cases; the negative pole may be placed over the lumbar spine and the positive upon the epigastrium or within the stomach itself. Repeated light contacts with the thermo-cautery over the spine and gastric region often aid greatly in effecting a cure.

BULIMIA.

This is a sensory gastric neurosis which may be defined as a pathologically increased sense of hunger. It is sometimes a symptom of certain organic affections of the stomach and sometimes an independent neurosis. It may be witnessed in some cases of exophthalmic goitre, chronic gastritis, gastric ulcer with hyperacidity, hysteria, pregnancy, hypochondriasis, psychoses, phthisis, and diabetes. It is occasionally a symptom of the presence of intestinal parasites, as of the tape-worm.

The intense hunger may appear periodically at intervals of perhaps a few weeks, or may come on shortly after nearly every regular meal. The attack is characterized by sensations of intense hunger accompanied by pallor and faintness which begin to pass away as soon as eating or drinking is commenced.

In the way of treatment the remote cause must be sought in every case and removed when possible. Remedies may also be administered with the intent of quieting the nervous centres presiding over the sensation of hunger. The bromides are useful for this purpose, as are valerian, zinc, and similar drugs. Opium and belladonna may likewise be administered. Arsenic may be of value, and the local effect of cocaine is sometimes beneficial.

ACORIA.

By acoria is indicated an absence of the sense of satisfaction after eating. It may be combined with bulimia or with anorexia. The patient, whether eating much or little, does not feel satisfied, and cannot tell by his sensations whether he has had enough.

The condition may be occasional only or persistent. Its occurrence is always combined with evidences of hysteria or neurasthenia. It is a distinct affection from bulimia, since in this latter the patient has intense hunger and eats largely until satisfied, while in acoria there may or may not be real hunger, but the sense of satisfaction never comes with eating.

Treatment must be directed to the nervous condition present. The ingestion of a definite amount of food should also be advised, in order that the stomach shall become accustomed to the presence of a certain fixed quantity.

NERVOUS ANOREXIA.

This is another sensory gastric neurosis which is characterized by extreme loss of appetite, lasting for weeks or months. There may even be an intense disgust for food. It is associated with hysteria, neurasthenia, or psychoses, while at the same time there is no evidence of any organic gastric affection. The anorexia may vary much in intensity. In some cases, especially in the insane, it may be so obdurate that there may be a fatal issue.

The treatment must be primarily that of the general cause, and secondarily must consist in the administration of the various tonics and stomachics. Orexin may be tried. Gentian and cinchona, alone or in combination with hydrochloric acid, are sometimes useful.

NERVOUS VOMITING.

Nervous vomiting is a condition which is of both frequent occurrence and of great difficulty to control. It does not depend upon any anatomical lesion of the stomach. It is a motor neurosis, the result of a disturbance of the nervous centres which control the act of vomiting. This disturbance may be direct or reflex. Among its causes are organic lesions of the kidneys, liver, brain, spinal cord, and uterus. It may occur, for example, in Bright's disease; renal calculi and abscesses; biliary calculi and abscesses of the liver; meningitis, and in tumors, abscesses, and hæmorrhages of the brain. Nervous vomiting is seen in the crises of tabes dorsalis, not infrequently accompanies uterine diseases, and is a well-known attendant upon pregnancy.

Apart from the existence of gross anatomical changes as causes of nervous vomiting, the disorder is seen in migraine and in sea-sickness. Neurasthenia is a cause of nervous vomiting, and hysteria is a very frequent one. Cases of periodic vomiting apparently of the nature of a neurosis have been described by Leyden and are not rare.

The symptoms of nervous vomiting differ from those of other forms in that there is seldom much nausea or retching. Ejection of the food is liable to occur soon after eating. In some hysterical cases apparently all food is vomited almost at once after it is taken, while in other cases only certain kinds of nourishment are rejected. Generally the patients suffer little disturbance of nutrition and the disease is not serious. This is particularly true of the hysterical form. Sometimes, however, much emaciation follows and death may occur.

The treatment is to be directed against the primal cause of the affection. In other respects it is to be carried out on the line already indicated for gastric neuroses.

NERVOUS ERUCTATION.

This is a motor neurosis which is entirely independent of abdominal fermentative changes, since the gas expelled is both odorless and tasteless. It is especially common in hysterical cases, although occasionally seen in neurasthenics as well. The gas probably consists, in part at least, of air which has been swallowed. There is present without much doubt an increased contractility of the stomach with a firm closure of the pyloric sphincter. As a result of this the air is expelled through the œsophagus in very large quantities. In some cases it comes from the œsophagus alone.

Regurgitation.

This consists of the involuntary rising of portions of food into the pharynx, or even into the mouth, whence they are expectorated. It is unattended by nausea. The causes and treatment are the same as those of rumination, from which it differs in that the food is not re-chewed and re-swallowed, but is spit out, if it rises as high as the mouth at all.

Rumination.

Rumination, or merycismus, is a rare neurosis which has long been recognized, and in which the patients regurgitate and remasticate and again swallow the food. The condition is oftenest seen in neurasthenic, hysterical, insane, or epileptic individuals, but not always in these alone. Instances of its inheritance are on record, as also of the acquiring of the habit by imitation. It may occur at any age and in any station of life. In most cases there is no anatomical alteration of the stomach or œsophagus or any permanent dilatation of the cardia. In a few instances dilatation of the stomach has been found and in a few others a sacculated distention of the œsophagus near its lower end. There may be a temporary relaxation of the cardia at the time regurgitation takes place. Changes in the gastric secretion have been found in some instances, but this varies in different cases.

The food is regurgitated at a variable time after eating. The various ingredients of the meal may be indistinguishably mingled or the taste of different articles may predominate in different mouthfuls regurgitated. The patient may have no control whatever over the rumination, or may be able to bring it on at will. The effort to suppress it is painful. Nutrition may be seriously interfered with, or the disease may last for years and affect the health not at all.

The best treatment is an earnest effort by the patient to resist the inclination and the prompt swallowing of the regurgitated masses without rechewing them. Any other palpable defect of digestion should be rectified and evidences of nervous disturbance combated.

Atony of the Stomach.

Atony of the stomach, or gastric insufficiency, as it is also called, is a neurosis to which reference is made elsewhere, since it may attend other pathological gastric conditions, whether organic or functional. Although so frequently associated with other states, it occurs also as a distinct affection independently of any secretory disturbance. It may occur as a primary neurosis whose seat is either in the central nervous system or in the stomach itself, and it may cause various dyspeptic symptoms, and even be finally productive of gastrectasia, as the result of the food remaining too long in the stomach. It is, indeed, one of the factors in the production of the dilatation, whatever the other causal conditions may be.

On the other hand, this motor neurosis may be secondary to functional, secretory, or organic affections of the viscus.

The treatment will be again spoken of under the heading of Dilatation of the Stomach. Such drugs as strychnine may be expected to increase the motor power, while at the same time such other methods will be required, both dietetic and medicinal, as will prevent too long continuance of food in the stomach. Lavage is a most important measure which is often required.

PNEUMATOSIS.

By this term is indicated a great distention of the stomach with gas, the result of a primary gastric neurosis. It may be either intermittent or persistent. A secondary distention may occur in various intestinal and gastric disorders, especially in gastrectasia, but this is not properly included here. The condition is supposed to be the result of a spasmodic contraction of both pyloric and cardiac sphincters.

The symptoms are a sensation of great distention in the epigastrium, and sometimes dyspnœa and præcordial pain. Relief follows the expulsion of the gas. The disease must be diagnosticated from distention of the transverse colon and from distention in organic gastric diseases.

Treatment is to be directed against the general nervous condition, and should include the use of bromides and drugs of this class.

PERISTALTIC UNREST.

Tormina ventriculi, or peristaltic unrest, first described by Kussmaul, is a condition of increased peristalsis of the stomach. The movements, which pass from left to right and are distinctly perceptible through the abdominal wall, are accompanied by gurgling sounds and by most annoying subjective sensations, although not by actual pain. The movements are, as a rule, most marked after eating. They may be increased by nervous excitement or may sometimes be made to cease entirely by the same means. The condition is oftenest associated with gastrectasia. It is generally seen combined with unrest of the intestines, particularly of the duodenum.

ANTIPERISTALTIC UNREST.

Antiperistaltic unrest has been described by Glax and others. In this state the peristaltic movements are reversed. When this is combined with an antiperistalsis of the intestines, as has sometimes been the case, scybalæ, or colored enemata, have been discharged from the mouth.

INCONTINENCE OF THE PYLORUS.

In this condition the pylorus remains open during the period of digestion, a time at which it should be closed. The disorder may, of course, result from carcinomatous or cicatricial involvement of the pylorus, causing rigidity without stenosis, or through some other mechanical interference. A purely neurotic form of incontinence—the only one to which reference is here made—has been described by Epstein, but is certainly rare. It has often seemed to me that this condition might be present in cases of nervous dyspepsia where, speedily after the ingestion of food, there develops intestinal peristalsis and unrest soon followed by imperfectly digested stools. Although the failure of the effort to distend the stomach artificially with gas may be looked upon as an indication of pyloric incontinence, yet this symptom is not very reliable, and there exist, in fact, no means of making a positive diagnosis.

Spasm of the Cardia.

This consists of a paroxysmal or persistent closure of the œsophageal opening of the stomach. Its cause is not known, except that it occurs in neuropathic persons and seems to attend such nervous disturbances as mental affections, neuralgia, palpitation of the heart, pregnancy, irritation from worms, and the like. It may also come on in cases of gastritis and of gastric cancer. In the paroxysmal form there is a painful sense of constriction in the region of the stomach with tympanitic distention; the condition disappearing with belching of gas in considerable quantities. In the chronic form there is especially marked an obstruction to deglutition, chiefly of solid food. The introduction of the stomach-tube shows the situation of the spasm to be at the cardia.

The diagnosis of the paroxysmal spasm is easy when the periodicity of the symptoms and the presence of general nervous phenomena are born in mind. The recognition of the chronic spasm is more difficult since the affection may simulate cancer of the cardia. The presence of general nervous symptoms and the fact that a thick sound passes with no more difficulty than a thin one point to the existence of cardiac spasm.

The treatment consists of the systematic employment of firm sounds of sufficiently large size and in the internal administration of nervines.

NON-INFLAMMATORY ORGANIC DISEASES OF THE STOMACH.

Simple Gastric Ulcer.

Definition.—A more or less round or oval, sharply cut, and usually single ulcer, which penetrates the mucous lining and sometimes the entire thickness of the gastric wall, and which is due to the action of the gastric juice upon a portion of the mucous membrane whose nutrition has been interfered with.

Synonyms.—Peptic ulcer; Ulcus ventriculi; Rodent ulcer; Penetrating ulcer; Chronic eroding gastric ulcer; Round ulcer of the stomach.

Etiology.—The disease is more frequent than the clinical symptoms would indicate, since autopsies reveal the presence of an open ulcer or of a scar in many instances in which no diagnosis was made during life. Statistics show that in about 5 per cent. of all autopsies there are ulcers, open or cicatrized, the latter being three times as common as the former. Females are attacked twice as often as males. The disease is commonest between the ages of twenty and forty years, although it has been observed occasionally in infants and children. Race appears to exert some influence, the lesion having been found in 13 per cent. in a series of autopsies in Copenhagen and in only 2.7 per cent. in a series in Berlin. These differences may depend upon the nature of the food eaten. Occupation is a powerful factor. Shoemakers, tailors, and weavers are prone to it, possibly as the result of direct injury to the stomach in the pursuit of their trades; and housemaids, especially female cooks, are likewise particularly liable to it. In states of impoverishment of blood, as in chlorosis and in anæmia of various kinds, ulcer is apt to develop. Insufficient food also appears to act as a cause. In a few instances extensive burns of the skin have been followed by gastric ulcer, while diseases of the heart and blood-vessels have repeatedly been associated with it.

Pathogenesis.—The method of production of a gastric ulcer has been much discussed, and many experiments upon animals have been made in the effort to understand it fully. As a result of all studies it would appear that at least two factors are required to produce the lesion, these being—(1) an interference with the nutrition of a small portion of the gastric mucous membrane, with either (2) the action upon this area of the hyperacid gastric juice, or (3) some alteration in the composition of the blood.

Virchow advocated the theory that the primary lesion was the plugging of an artery of the part by a thrombus or embolus, as a result of which an infarct was produced. It is in this way that heart disease would take its part in the formation of the gastric ulcer. So also other lesions of the gastric mucous membrane may be the primary cause of an ulcer, among them being mechanical or thermal irritation, circumscribed stasis in the circulation, local hæmorrhages from congestion, and the like. In the same category—viz. that of local interference with nutrition—may be placed the production of gastric ulcer by microbes. Several investigators have claimed this microbic origin, and have succeeded in producing a lesion in animals by the subcutaneous injection of cultures. Either an embolus or a direct invasion by the microbes is said to result.

None of these causes alone, however, can produce a persistent gastric ulcer, for experience has shown that ulcers artificially produced in animals heal with remarkable rapidity, while, on the other hand, so many individuals are exposed to lesions from hot food, blows on the epigastrium, and the like that, could these suffice to produce the disease, ulcers should be many times more frequent than experience shows them to be.

The theory was advanced by Pavy that for the production of gastric ulcer a diminished alkalinity of the blood was necessary, as a result of which the gastric juice was permitted to digest portions of the mucous membrane. There are serious objections to this view, although the fact that anæmia and chlorosis both possess a diminished alkalinity and predispose to ulcer indicates that it may possibly serve as a partial explanation. At any rate, some change in the normal constitution of the blood, as a result of which the cells are, perhaps, insufficiently nourished, is certainly a predisposing factor.

More recent studies, among which those of Riegel may perhaps be especially mentioned, have discovered the important fact that in very many cases of gastric ulcer there is a decided hyperacidity or hypersecretion of the gastric juice. The claim was then made that hyperacidity was an essential factor in the production of ulcer. This does not, however, appear to be the case since ulcer may be present without this hyperacidity or may fail to develop even when it exists.

The conclusion may be drawn that the first and the essential step in the formation of a gastric ulcer probably is the production of some distinct localized lesion of the gastric mucous membrane in some of the ways mentioned. Upon the spot thus injured a hyperacid gastric secretion constantly acts, whether or not the condition of the blood is also altered; while in other cases the altered character of the blood, as seen in chlorosis and the like, permits even a normally acid gastric juice to act upon the mucous membrane in a destructive manner not otherwise possible. In many cases probably both secondary factors are simultaneously in action.

Pathology.—Though sometimes multiple, the ulcer is single in the great majority of cases. In one case reported by Berthold 34 ulcers were found. The lesion is usually situated on the posterior wall of the pyloric portion of the stomach near the lesser curvature. It varies in diameter from half an inch to two inches, although it may be larger or much smaller than these dimensions. The shape of the ulcer is usually round or oval, although it is sometimes irregular. It is generally somewhat funnel-form, with sharply-cut edges, giving at first the well known "punched-out" appearance; but as it grows older the walls become indurated and thickened. The floor of the ulcer, though usually smooth, varies in character and appearance with the depth to which the destruction has penetrated. It may even consist of some tissue external to the stomach if perforation and adhesive inflammation have taken place.

Microscopic examination shows that the process is a necrosis and not a true ulceration. The parts surrounding the ulcer are made of broken-down red blood-corpuscles, granular material and cells which stain but poorly, together with fibrin, hyaline masses and scattered red blood-cells in the parts a little more removed. As the ulcer grows older a small-celled infiltration takes place about it, showing a tendency to repair; and as healing takes place fibrous tissue forms in the walls and floor, and, contracting, leaves a smooth, stellate, white scar with the mucous membrane puckered about it. When the patches are small, the edges of the mucous membrane may be so approximated by the contraction that the scar is scarcely, if at all, visible. If the ulcer has been large, the cicatricial contraction may cause various deformities. Thus, if near the pylorus, stenosis may be produced with consequent gastrectasis, or an hour-glass contraction may result from the cicatrization of a series of ulcers extending around the stomach in the form of a girdle.

Sometimes there is no tendency to healing, but a steadily progressing corrosion goes on until the coats of the stomach are perforated. A circumscribed adhesive peritonitis often develops in advance of the necrotic process, attaching the stomach to surrounding organs and preventing immediate rupture of the ulcer into the peritoneal cavity. Owing to the usual position of the ulcer these adhesions are commonest with the pancreas and the left lobe of the liver.

The results of perforation are very varied. Considerable portions of the liver or spleen, or, to a less extent, of the pancreas are sometimes eaten away and an intraperitoneal abscess thus formed. Direct opening into the peritoneal cavity at once causes death from shock or from an intense and fatal peritonitis. If the opening be through the posterior wall, it enters the lesser peritoneum, and may thus produce a subphrenic pyopneumothorax. Perforation may take place into the pleura, the lung, the pericardium, the gall-bladder, the left ventricle, the colon or the small intestine, or through the skin forming a fistula.

Very often the necrosis extends through the walls of some blood-vessel and produces hæmorrhage, which may be quickly fatal. The commonest cause of such fatal hæmorrhage is erosion of the splenic artery by an ulcer in the posterior wall of the stomach. Cases of ulcer which have run a chronic course are most liable to suffer from severe hæmorrhage.

A considerable number of cases exhibit at the autopsy changes in the blood-vessels. Embolism in the arteries supplying the affected parts has been found in several instances. Thrombosis of arteries or veins has also been observed, as has diffuse endarteritis and small aneurisms in the floor of the ulcers. A varicose condition of the veins of the stomach and hyaline degeneration of the arteries have each been reported.

Symptomatology.—The symptoms are somewhat variable, since an ulcer may run its course and heal without presenting any clinical evidences whatever, or at least none at all characteristic, the lesion being discovered only accidentally post-mortem. In other cases the symptoms may be very vague, and only those which could be attributed to ordinary dyspepsia. In still other cases a profuse hæmorrhage or the evidence of perforation into the peritoneal cavity is the first sign of the existence of an ulcer, and in still others symptoms more or less characteristic exist.

The cardinal symptoms are peculiar pain in the epigastrium, tenderness on pressure, hæmorrhage, vomiting and other evidences of dyspepsia of varying degrees of severity.

Pain is one of the most constant and characteristic symptoms of the disease, and is rarely continuously absent. It may be only moderate in severity, and be but a gnawing, burning sensation which comes on during the night or at other times when the stomach is empty, and which occasionally is dissipated by taking food. A more characteristic condition, however, is the development of paroxysms of intense pain which appear a few minutes after eating or not for a couple of hours. Too hot, too cold or sour articles give more pain than unirritating ones. The pain is localized in a small spot in the epigastrium and is oftenest felt at or just below the xiphoid cartilage.

Very frequently severe diffuse gastralgia is combined with the local suffering, and the pain radiates to the sides and shoots through to the back. Here it may be experienced in the right shoulder or between the scapulæ, and sometimes lower than this, and is often a little to the left of the spinal column. The attacks of pain may come on frequently during weeks or may cease entirely for a longer period of time. They are usually made worse by pressure, though sometimes eased in that way, and are generally relieved by rest in bed. The pain may at times be brought on by excitement, exposure to cold and fatigue. In the intervals between the attacks there is often experienced some discomfort or even a dull pain in the epigastrium. There may be also a sense of distention and of oppression after eating, this being due to the attendant gastritis.

Tenderness on pressure is a very common symptom. The spot of tenderness is usually quite small, and is oftenest situated just below the xiphoid cartilage. Patients are unable to wear any tight clothes about the waist. The examination must be conducted with care, for not only is it painful to the patient, but also not devoid of danger. If the ulcer be old and its edges thickened, a sense of increased resistance over a small area or much more rarely a hard mass may be felt during the examination. The presence of tenderness is not absolutely diagnostic, since in some individuals the epigastrium is constantly tender on pressure.

Hæmorrhage occurs in a proportion of cases which has been variously estimated as from one-fourth to four-fifths. As an average it may be stated that it occurs in about one-third of the cases. The amount of blood lost may be slight or very large. Generally it is sufficient in quantity to be vomited while still of a bright red color, for the presence of a large amount of blood in the stomach usually induces vomiting. If the hæmorrhage be small, or if larger amounts have remained some time in the stomach, the blood may be vomited in an altered form and may be in reddish-brown or even dark-brown masses. Sometimes all the blood is passed by the stools, and this may occur rarely even when the hæmorrhage has been large. In most cases, however, if much blood be vomited, some is passed by stool as well. The blood passed from the bowel is entirely altered in character, having become tarry in consistence and color.

A hæmorrhage may be followed by syncope or collapse, or even, exceptionally, by sudden death. The development of syncope stops the bleeding temporarily, but it recurs shortly, and the process may continue thus interruptedly during several days until a very high degree of anæmia with its attendant symptoms is produced, or until even death takes place. If the hæmorrhage cease and the symptoms of anæmia gradually disappear, there is still the great probability that another similar attack of hæmatemesis will occur after an interval more or less prolonged, although recovery sometimes takes place after a single attack. The interval between the attacks may be but a few days or may be some years. Other symptoms of ulcer, and especially the pain, are sometimes relieved temporarily by the occurrence of hæmorrhage.

Of the digestive symptoms present in gastric ulcer, the most common is vomiting, although it is not, of course, diagnostic of the disease. It occurs most frequently after taking food, and is often such a troublesome symptom that the nutrition of the patient is seriously interfered with. Frequently every article of food taken is vomited shortly afterward. The ejected matter generally contains a large amount of hydrochloric acid, and evidences of fermentation are usually absent. The vomiting is the result chiefly of a direct irritation of the ulcer by the ingested food.

The other dyspeptic symptoms, often best seen in the early stages of ulcer before they are masked by the graver ones of the fully-developed disease, are disturbances of appetite, indefinite sensations of gastric discomfort, and occasionally evidences of chronic gastritis. The tongue is generally clean or but slightly coated, and is sometimes unusually red. Constipation is frequent.

Among the general symptoms often present are loss of weight, which results chiefly from inability to take and digest sufficient nourishment, vertigo, headache, depression of spirits, oppression in breathing, and other symptoms dependent upon dyspepsia. Fever is not present in gastric ulcer excepting from some complicating inflammatory process. Amenorrhœa is a very common symptom in women, and the frequency of anæmia has already been referred to.

Perforation of an ulcer into the general peritoneal cavity takes place in about 6.5 per cent. of all cases, according to the statistics collected by Welch. It may be brought on by some movement or by taking food, and is much commoner in women than in men. Its occurrence is announced by the development of intense pain. Although this pain is generally first felt in the epigastrium, extending thence over the abdomen, it is sometimes referred entirely to other parts. Then develop collapse and, later, distention of the abdomen, great sensitiveness to touch, small, rapid pulse, Hippocratic face, shallow respiration, and other characteristic symptoms of perforative peritonitis.

Complications and Sequels.—Chronic gastritis is a frequent complication to which reference has already been made. Perforation has already been referred to. Pylephlebitis is an important complication, leading generally to abscess of the liver. Parotitis, presumably of septic character, may develop. Chronic peritonitis may also occur. Cancer may develop in the edges of the ulcer. Certain of the symptoms, as amenorrhœa and perforation, might with equal propriety be regarded as complications.

Of sequelæ the most important is dilatation of the stomach consecutive to pyloric stenosis, the result of cicatricial contraction of the ulcer. Cirrhosis of the stomach sometimes follows or attends ulcer.

Diagnosis.—The diagnosis is sometimes easy, sometimes impossible. It is based upon the occurrence of pain, vomiting and hæmorrhage. When all the symptoms occur conjointly, the disease can scarcely be mistaken, but where this is not the case, and especially where hæmorrhage is absent, the diagnosis must remain somewhat uncertain. The points of distinction between ulcer and cancer of the stomach will be considered later.

In the absence of hæmorrhage it is often exceedingly difficult to distinguish between gastric ulcer and nervous disorders of the stomach. Nervous dyspepsia rarely exhibits the intense epigastric pain and tenderness, and vomiting is not common. It likewise occurs in conjunction with other nervous symptoms. Hyperacidity of the gastric juice may exist in each condition. Nervous vomiting occurs in hysterical or neurasthenic cases, or associated reflexly with some definite remote organic lesion. There is also less pain and tenderness.

Usually neither of these diseases can be confounded with gastric ulcer, unless the symptoms of the latter be very ill defined. Nevertheless, the diagnosis in hysterical cases is occasionally wellnigh impossible.

Purely functional gastralgia often simulates gastric ulcer very closely. In the former, however, there is not the localized pain and tenderness characteristic of ulcer, and the more diffuse pain is generally lessened by pressure. Again, the ingestion of food usually brings on an attack of pain in ulcer, while it relieves the pain of gastralgia. To this rule, however, there are exceptions. Vomiting is not so frequent in gastralgia, and when present does not relieve pain as it does in ulcer. The general nutrition may be well preserved in either disease, but is more apt to suffer in ulcer. In ulcer some pain or some sensation of discomfort is liable to persist between the paroxsyms, while in gastralgia this is not the case. The accompanying symptoms and the general state of the patient are frequently an index of the nature of the disease, since in gastralgia there are usually present evidences of hysteria, neurasthenia, or neuralgic affections, while in ulcer there are none of these, but chlorosis or anæmia are common in female patients. Finally, the therapeutic test is of value, for the treatment that is of avail in one is useless in the other.

Duodenal ulcer simulates gastric ulcer so exactly in many cases that the diagnosis is difficult or impossible. Sudden intestinal hæmorrhage following severe gastralgia points toward a duodenal ulcer, although not positive proof of it. The character and situation of the pain are much the same in the two diseases.

Biliary colic may sometimes resemble gastric ulcer in so far as the pain is concerned, but its situation, onset, and termination, the position of the pain toward the right, the occurrence of jaundice, and the occasional enlargement of the liver and gall-bladder, generally render the diagnosis easy.

Chronic gastritis has few points of resemblance to ulcer. There are no gastralgic attacks and localized pain, while there are flatulent distention, eructations, and similar characteristic symptoms.

Prognosis and Course.—The course of the disease varies greatly. Some cases are very acute, fatal perforation or hæmorrhage taking place early, but the great majority run a chronic course. Welch describes as follows a number of clinical forms illustrating the course of the disease in different cases:

"1. Latent ulcers, with entire absence of symptoms, and revealed as open ulcers or as cicatrices at the autopsy.

"2. Acute perforating ulcers. With or without a period of brief gastric disturbance perforation occurs and causes speedy death.

"3. Acute hæmorrhagic form of gastric ulcer. After a latent or a brief course of the ulcer profuse gastrorrhagia occurs, which may terminate fatally or may be followed by the symptoms of chronic ulcer.

"4. Gastralgic-dyspeptic form. In this, which is the most common form of gastric ulcer, gastralgia, dyspepsia and vomiting are the symptoms. Sometimes one of the symptoms predominates greatly over the others, so that Lebert distinguishes separately a gastralgic, a dyspeptic, and a vomitive variety. Gastralgia is the most frequent symptom.

"5. Chronic hæmorrhagic form. Gastrorrhagia is a marked symptom, and occurs usually in combination with the symptoms just mentioned.

"6. Cachectic form. This usually corresponds only to the final stage of one of the preceding forms, but the cachexia may develop so rapidly and become so marked that the course of the disease closely resembles that of gastric cancer.

"7. Recurrent form. In this the symptoms of gastric ulcer disappear, and then follow intervals, often of considerable duration, in which there is apparent cure, but the symptoms return, especially after some indiscretion in the mode of living. This intermittent course may continue for many years. In these cases it is probable either that fresh ulcers form or that the cicatrix of an old ulcer becomes ulcerated.

"8. Stenotic form. By the formation of cicatricial tissue in and around the ulcer the pyloric orifice becomes obstructed and the symptoms of dilatation of the stomach develop."

The duration varies much. In some cases the disease lasts from twenty to thirty years, but it is a question in these whether the original ulcer persisted or whether new ones had formed. The average duration is from three to five years. An ulcer once healed sometimes reopens, so that relapses may occur either in this way or by the formation of new lesions. Death may result from perforation, exhaustion, hæmorrhage, chronic anæmia or some complication. Death from perforation into the peritoneal cavity takes place, as already stated, in about 6.5 per cent. of all cases. Death from hæmorrhage occurs in from 3 to 5 per cent. of all cases. Probably 15 per cent. of all cases of gastric ulcer are fatal.

The prognosis in any individual case is difficult to make, as the disease is so treacherous. Naturally, the recent cases are more amenable to treatment than those in which the ulcer is already old and indurated. The occurrence of severe hæmorrhage makes the prognosis grave, since, even if not at once fatal, the accident is so liable to recur. It also indicates that the ulcer is already deep. Perforation is almost invariably fatal.

Treatment.—This consists primarily in the employment of such measures as will favor the healing of the ulcer, and secondarily in such symptomatic treatment as indications call for. As soon as the diagnosis is made, or if grave suspicion of the existence of ulcer is justified, the patient should be confined absolutely to bed. For the first few days, at least, alimentation should be entirely rectal. For this purpose peptonized foods are to be selected, or such substances as defibrinated blood or other blood preparations. Where vomiting is obstinate it may be necessary to limit feeding to the rectal method for a considerable time. To relieve thirst small pieces of ice may occasionally be sucked or swallowed.

Nourishment by the mouth may generally be commenced after a few days. It should consist of the lightest and most digestible food given frequently in small quantities. Milk, plain or peptonized, buttermilk, prepared beef foods of various kinds, egg albumin, broths and the like are suitable. Considerably later, as the case improves, the dietary may be extended. This should not be done before three or four weeks. Sweetbreads, starchy substances, white meat, of chicken and eggs may now be tried. Meantime the use of Carlsbad water, recommended by Ziemssen, may be commenced. The object of this is to neutralize the acidity of the stomach and to wash its contents into the intestine, while at the same time its laxative quality relieves constipation. Half a pint to a pint of the water taken hot should be swallowed slowly once a day in the early morning. In place of the water a mixture may be made of sulphate of sodium, five ounces ; bicarbonate of sodium, two ounces ; chloride of sodium, one ounce. Of this a heaped teaspoonful may be taken in a pint of warm water.

When vomiting is very obstinate and all food is rejected, alimentation must be entirely rectal ; an application of the thermo-cautery should be promptly made at the epigastrium, and the use of nitrate of silver should be instituted. This drug, which is by far the most valuable remedy in the treatment of ulcer, may be given in pill form in the dose of one quarter of a grain associated with opium. Other measures to relieve vomiting are the swallowing of ice, the administration of bismuth, bismuth and morphine, hydrocyanic acid, chloroform, champagne, and carbonated water. Morphine hypodermically administered is often very useful. Lavage of the stomach may be found useful in severe or persistent cases, warm alkaline water being employed twice a day. The method is not without some danger, yet the risk is slight and the advantages often great. Sometimes food may be introduced into the stomach through the tube and is often retained only when given in this way. The tube must not be used if hæmorrhage has recently occurred. It is best to reserve lavage for cases in which other treatment for vomiting is futile.

For the relief of pain it is at times necessary to use morphine hypodermically, but the great danger of the formation of a habit must not be forgotten. Hoffman's anodyne or chloroform sometimes relieves. Ice over the epigastrium is of service in some cases, while in others warm applications or mustard plasters are to be preferred.

For the treatment of hæmorrhage nothing is so useful as the swallowing and the local application of ice, combined with the hypodermic administration of morphine and ergot. No food whatever should be taken by the mouth. Threatening syncope should not be too quickly treated by stimulation, since during the depressed state of the circulation bleeding may be stopped by the formation of a thrombus. If necessary the heart may be stimulated by hypodermic injections of ether, camphor, or alcohol. Transfusion of blood or intravenous injection of salt solution may be made if death be impending. If done immediately after a hæmorrhage, however, it is apt to cause the renewal of this as the result of the increased blood-pressure.

If perforation take place, the only method of medical treatment available is the administration of large doses of opium and, if necessary, stimulants, and the local application of hot fomentations or of cold. The question of operative interference may also be entertained.

Treatment directed to the general health is often indicated as the attack passes off. For this purpose iron or arsenic is often useful, since the patients are so frequently anæmic. Some unirritating preparation of iron, such as the albuminate, citrate, or peptonate, is to be selected.

OTHER ULCERATIONS OF THE STOMACH.

In addition to the form of gastric ulcer just described certain other varieties exist. They need little more than mention in this connection.

Cancerous ulceration will presently be referred to. Hæmorrhagic erosions are quite common. They have no special clinical significance, and may be produced in the course of a variety of morbid processes. The same may be said of the follicular ulcerations which have been described.

In addition to what has already been said of the ulcers seen in toxic, phlegmonous, parasitic, membranous, and chronic gastritis, notice must be taken of the tubercular and syphilitic ulceration and of that of typhoid fever. Typical typhoid ulcers have very occasionally been found in the stomach. They produce no characterisic symptoms, but have been known to cause hæmorrhage or perforation.

Tubercular ulceration of the stomach is uncommon. It has been discovered only in association with tuberculosis of other parts, and especially with ulceration of the intestine or with tubercular lymphatic glands. The ulcers are pale and with thickened edges. Nodules containing giant-cells, bacilli, and perhaps cheesy matter are present in the floors and walls. There are no characteristic symptoms, but hæmatemesis or perforation has been reported.

Syphilitic ulceration of the stomach has been much discussed. Such ulcers, if present, would have no characteristic anatomical features. An ulcer primarily due to syphilis would probably have the appearance and run the course of a simple gastric ulcer, and could not be distinguished from it. The existence of a syphilitic ulcer would simply mean that syphilis is to be included among the causes of simple gastric ulcer. There seems, however, to be no method of positively determining whether the association of gastric ulcer and of constitutional syphilis in an individual is simply a coincidence or whether there is some etiological relation between them.

CANCER OF THE STOMACH.

Definition.—A morbid growth of the gastric walls composed of connective-tissue alveoli containing epithelioid cells. It usually occurs in advanced life, spreads into the surrounding tissues, produces characteristic symptoms, and always terminates fatally.

SYNONYMS.—Among the various names applied to the different varieties of cancer are hard, scirrhous, soft, encephaloid, medullary, colloid, gelatinous, and cylindrical-celled epithelioma.

Etiology.—The frequency of cancer of the stomach can be only approximately determined, since statistics vary between 0.6 to 3.5 per cent. As an average, Welch estimates that cancer of the stomach is the cause of death in 1 per cent. of all persons dying after the age of twenty years. Next to primary cancer of the uterus, that of the stomach is the most frequent form, and the latter affection is considered by some writers to lead in point of frequency.

The actual cause of cancer of the stomach, as of cancer of any part of the body, is not understood. Certain predisposing causes, however, are recognized. Race and climate have been claimed to exert a predisposing influence. The disease is said to be rarer in South America, India, Egypt, and Turkey than in some other countries, less common in the United States than in Europe, and very rare among negroes in Africa. It is very uncertain, however, whether these claims will bear the test of a more extended statistical investigation.

Sex does not appear to exert any material influence, although in my personal experience the disease has been decidedly more common in men. Age is a most important factor. According to the statistics collected in the valuable paper of Welch in the *System of Practical Medicine by American Authors*—to which I am indebted for much of my statistical data—three-fourths of all cases occur between forty and seventy years of age, with a somewhat greater number between fifty and sixty than in any other decade. The disease is much less often seen between twenty and thirty, very seldom between ten and twenty, and with the greatest rarity at an age of less than ten years.

The influence of heredity has been a much disputed matter. It probably does not exert the power which has been attributed to it, but seems undoubtedly to act in some cases as a predisposing cause.

Cancer may rarely develop in the indurated walls of an old gastric ulcer. Chronic gastritis and traumatism of the stomach, including the action of corrosive substances and of blows, have frequently been assigned as causes of the disease, but their real influence is doubtful. The station in life, occupation, general health, condition of the emotions, and the existence of previous disease of any sort appear to be totally without influence.

Pathology.—The several varieties of cancer which occur as primary growths in the stomach are scirrhous, encephaloid, colloid, and cylindrical-celled epithelioma. Various intermediate forms exist between these, so that it is frequently difficult to determine to just which class a growth belongs. Scirrhus is usually stated to be most common and colloid the least so, but the statistics collected by Welch make the soft cancer, including encephaloid and epithelioma, the most frequent.

Regarding the part of the stomach involved, the pyloric region is attacked in about one-half of all cases, while the fundus is least often diseased. Welch submits the following table as the result of the analysis of 1300 cases :

Pyloric region.	Lesser curvature.	Cardia.	Posterier wall.	The whole or the greater part of the stomach.	Multiple tumors.	Greater curvature.	Anterior wall.	Fundus.
791	148	104	68	61	45	34	30	19
60.8%	11.4%	8%	5.2%	4.7%	3.5%	2.6%	2.3%	1.5%

Cancer begins in the mucous membrane, and soon involves the whole thickness of the walls. It may spread rapidly in extent or, as in the case of scirrhus, may grow principally in depth and height. Sometimes a cancer forms a ring around the stomach, particularly in the region of the pylorus. The portion of the growth which projects into the cavity of the stomach may be broad, flat, or cauliflower-like. Ulceration may take place in any form of cancer, and sloughing masses may separate. In this way hæmorrhage may be produced. Ulceration is commonest in encephaloid and cylindrical-celled epithelioma, less so in scirrhus, and least frequent in colloid. Perforation, too, not unfrequently occurs, but opening into the peritoneum is very frequently hindered by the advancing growth of the tumor, which usually forms adhesions with the adjacent parts. Perforation into the peritoneum is estimated by Brinton to occur in about 4 per cent. of all cases. It may also take place into the transverse colon and be followed by vomiting of fæcal matter and the passage of undigested food in the stools. Very rarely the abdominal wall has been perforated, or fistulæ have formed into the lungs, pleura, and small intestine.

Scirrhous cancer may appear either as a small, circumscribed tumor or as a diffuse thickening of the walls of the stomach. It is oftenest seen at the pylorus, and may produce stricture. It ulcerates only superficially. It consists of a large amount of dense fibrous tissue, with relatively few and small alveoli.

Encephaloid cancer grows rapidly, soon invades all the coats of the stomach, and forms large, irregular masses which project into its cavity and tend to ulcerate easily. It is soft and of a grayish-white or reddish-white color. Metastasis is more frequent in this variety than in any other. Microscopically, there is but a small amount of connective tissue as compared with the cellular element.

Cylindrical-celled epithelioma is occasionally hard like a scirrhus, but generally resembles more closely an encephaloid, although it is somewhat firmer, particularly at its edges. It also has a proneness to ulcerate and to form metastases. Microscopically, it resembles the structure of tubular glands, the alveoli being lined by columnar cells. Very frequently a portion of the growth exhibits the histological character of an ordinary cancer. Cylindrical-celled epithelioma is very apt to exhibit cysts which contain mucus.

Colloid cancer shows itself as an extensive, rather uniform infiltration and thickening of all the layers of the wall of the stomach. It spreads very often to adjacent structures, especially the peritoneum, though it rarely produces actual metastasis. It exhibits trabeculæ of white connective tissue forming large alveoli filled with a peculiar transparent, gelatinous substance. Ulceration may occur, but it is usually superficial. The colloid metamorphosis may attack any form of gastric cancer, but especially the cylindrical-celled epithelioma. Microscopically, few epithelial cells are discoverable.

All that has been said refers to primary growths of the stomach. Only rarely does cancer appear here secondarily to a growth elsewhere. On the other hand, cancerous growths in various other parts of the body, or even in another part of the stomach, secondary to a gastric cancer, are exceedingly common. They oftenest occur in the lymphatic glands in the neighborhood of the stomach, and next in frequency in the liver. The liver is involved in about one-third of all cases. General involvement of the lymphatic glands in different parts of the body is not so common as in cancer of some other parts, notably the breast.

Various alterations in the shape and position of the stomach may be produced mechanically by cancer. The organ may grow very much smaller as a result of contraction when the growth has involved much of the wall. Atrophic shrinking of the stomach will also follow obstruction of the cardia, while dilatation may result from pyloric stenosis. Various distortions and constrictions may be produced by a cancer, and the weight of the growth may make remarkable alterations in the position of the whole stomach, which has even been found in the pelvis.

Secondary to cancer there are very liable to be present the lesions of chronic gastritis. Hypertrophy of portions of the mucous membrane or of the other layers also of the gastric wall may occur. Atrophy of the mucous membrane is stated to be occasionally induced by cancer.

Symptomatology.—The symptoms of cancer of the stomach are in some cases so obscure that the disease is never recognized at all or only shortly before death. There may, indeed, be no symptoms except those of asthenia, and well developed cancer may even be found in individuals dying suddenly from other causes in the midst of apparently good health and strength. In other cases the symptoms are very typical. Between these two extremes can exist all grades of development of characteristic symptoms. Sometimes one symptom predominates and sometimes another, and sometimes those of a complication, particularly cancer of the liver or peritoneum, greatly overshadow those belonging to the stomach itself.

The earliest symptoms are impairment of appetite, disturbance of digestion, a tendency to nausea and constipation, and some abdominal discomfort. At the same time there is very evident loss of strength and of weight beyond what the symptoms seem to account for. The abdominal discomfort now becomes actual pain, made worse by taking food. Vomiting also begins and becomes a troublesome symptom. Altered blood may be vomited.

The patient by this time is growing quite emaciated, and is assuming a decidedly anæmic, cachectic appearance. Examination of the abdomen will generally discover a hard tumor. The course of the disease continues unabated, although perhaps with temporary improvement of one or another symptom, until death takes place.

It is necessary to consider the individual symptoms somewhat more in detail. Loss of appetite is generally one of the earliest and most constant symptoms. Sometimes it is only with reference to certain articles of food, especially meat. A decided degree of capriciousness of appetite is noticeable, or occasionally there may be no loss of appetite whatever at any time during the disease. The tongue is very often coated considerably, but may be moist and clean. The taste is impaired ; salivation may occasionally be present.

Pain is an early symptom and a very frequent one. It is usually located in the epigastrium, but may be felt between the scapulæ, in the shoulders, in the loins or in the hypochondria. In cancer of the cardia the pain may be referred to the xiphoid cartilage, and in cancer of the lesser curvature to the interscapular region. As a rule, however, the locality of the pain is no indication of the exact seat of the growth.

The pain varies greatly in quality and in intensity. It is burning, gnawing, lancinating, and the like, and only occasionally comes in the form of severe gastralgia. It is generally made worse by taking food, although this may not become evident until a considerable time after a meal. This symptom also is not so constant as in gastric ulcer. Pain may occur independently of the presence of food in the stomach. Tenderness on pressure over the epigastrium or over the seat of the growth is usual.

In many cases pain, as already stated, may be absent or inconsiderable throughout the whole course of the disease. This is particularly true in individuals of advanced age. In the majority of cases, however, more or less pain is present.

Various other gastric symptoms are prominent. In the early stages there are evidences of slight indigestion, and as the disease advances these often become more marked. There are then discomfort after eating, distention, nausea, eructation of gas or of sour or offensive liquid. Nausea is more frequent than in the case of simple ulcer of the stomach, and hiccup is occasionally very annoying. Often the symptoms of severe indigestion characteristic of dilatation of the stomach are produced, since this condition is the direct result of obstruction of the pylorus by a cancerous growth.

One of the most important and most constant of gastric symptoms is vomiting, estimated to be present in 80 to 88 per cent. of the cases. It is sometimes, but not usually, one of the early symptoms. It takes place oftenest in cases in which the pylorus or the cardia is involved, but particularly the former. When the cancer is seated in other parts of the stomach, vomiting is generally absent or only occasionally occurs.

When it is seated at the pyloric opening, vomiting usually takes place some hours after the meal, although there are marked exceptions to this rule. As the stomach dilates, vomiting may become less frequent but more copious. Several days may elapse, and then there may be ejected very large amounts of gastric contents of the nature characteristic of gastrectasia, and which is described elsewhere.

When the cancer involves the cardiac opening, vomiting is apt to occur very soon after taking food. If the food be entirely unchanged in character and of alkaline reaction, it indicates that it has not entered the stomach at all. The vomiting in such cases is probably due to a spasm of the œsophagus, if the passage of a sound reveals no stricture. Dysphagia is also often present in cancer of the cardia. It may appear either as an early or a late symptom.

Vomiting and other symptoms of stenosis may disappear through the breaking down of a tumor and the removal of the obstructing masses. Small cancerous masses have very occasionally been discovered after the use of the stomach-tube, either adherent to it or present in the gastric contents which have been removed. Such masses are only rarely if ever observed in the vomited matter.

Hæmorrhage from the stomach is of common occurrence, although the amount lost is rarely large. The blood is generally altered in appearance and of a black or dark-brown color, forming the well-known "coffee-ground" vomit. This form of black vomit occurs in about one-half of the cases. That the material is really blood can be determined by the microscope, which reveals broken or decolorized red blood-cells and masses of blood-pigment. Where there is still doubt, hæmin crystals may be produced in the usual manner with acetic acid or the spectroscope may be employed. Emesis of large amounts of blood still presenting its normal appearance, or at most somewhat darkened in color, is not common. Lebert states that it occurs in about 12 per cent. of cases. It is sometimes sufficient in amount to cause death. Profuse hæmorrhage is most liable to take place in the later stages of the disease and from growths situated at the pylorus or on the lesser curvature.

In recent years a great deal has been written regarding the absence of free hydrochloric acid from the secretion of a carcinomatous stomach. It was at first claimed that this absence was persistent in all cases of carcinoma, and very rarely so in any other gastric disease. The whole subject is not yet entirely settled, but our present knowledge of the matter would seem to be represented by the statement that free hydrochloric acid is constantly absent in the majority of cases of cancer, at least so far as can be detected by the color tests. Repeated examinations should be made before a decision is reached in any case. Hydrochloric acid may be absent in other conditions as well as this, as, for example, in atrophy of the gastric mucous membrane and in chronic gastritis, although not so uniformly as in cancer. Its absence, therefore, is not of itself diagnostic of cancer, although in combination with other symptoms it is very suggestive. On the other hand, the presence of free hydrochloric acid is a very strong indication that the case is not one of cancer, for its occurrence in this disease is very uncommon, although it does sometimes happen.

The most valuable symptom of cancer of the stomach is the discovery by palpation of a tumor in the epigastrium. This can be made in about 80 per cent. of all cases, although usually not until the disease is well advanced. Only certain portions of the stomach, however, are accessible to the examination. Reference to the brief description of the position of the stomach given at the beginning of the section upon its diseases shows that a large portion of the organ lies beneath the ribs in the left hypochondrium and cannot be reached by palpation, and that still another portion is covered by the left lobe of the liver. Tumors, unless very large, can only be felt when situated on the anterior wall, on the exposed part of the greater curvature or at the pylorus.

Those at the pylorus can usually be felt even when overlaid by the liver. Tumors of the cardia cannot be detected at all, and those in the fundus, the lesser curvature, or on the posterior wall only when very large or if the stomach has been displaced.

The tumor is usually felt either in the epigastrium to the right of the median line or in the umbilical region. Sometimes it is in the right hypochondrium, or even as low as the iliac region, and occasionally it is felt close under the ribs to the left of the median line. It is generally firm, hard, nodular, and tender. Occasionally, however, it is smooth. If at the pylorus, it can sometimes be grasped between the fingers, and it often transmits the pulsation of the abdominal aorta beneath it. It is often immovable as a result of adhesions, and although movable by the hand it is but little affected by the respiratory movements of the diaphragm unless it be adherent to it or to the liver. There are, however, numerous exceptions to the rule. Sometimes deep inspirations will make discoverable a tumor which could not be detected before. At other times the emptying of the stomach by the tube and its inflation with gas or air will be followed by the same result. By the latter method a tumor of the posterior wall is made to disappear.

The percussion-note over a gastric tumor is usually that of dull tympany. Sometimes a sensation of abnormal resistance is all that can be discovered when the growth is a diffuse infiltration of the gastric walls. The situation of the tumor may change with alterations in the fulness of the stomach and intestine. In performing palpation the patient should be made to lie upon the back with the knees drawn up and the abdominal wall as relaxed as possible. It is sometimes also well to examine the patient in the knee-breast position or when standing erect.

It is often difficult to determine whether a tumor discovered really belongs to the stomach or to some other structure. Hard fæcal masses in the colon may simulate a cancer of the stomach. The importance of giving a laxative before making a positive diagnosis must never be forgotten. Dilatation of the stomach is an indication that the growth is situated in the pylorus, and not in the liver, omentum, or intestine; and failure to distend the stomach by effervescent powders points to pyloric insufficiency, perhaps the result of pyloric cancer, although not necessarily so. It is also often difficult to determine whether a tumor felt is a carcinoma or an induration of the stomach of some other nature. The above remarks indicate sufficiently the necessity for caution, at least in the earlier stages of the case, in deciding as to the actual existence of tumor, and even more so as to its exact nature and relations.

The bowels are generally affected in some way in gastric cancer. Constipation is usual, but occasional diarrhœa often occurs, and is sometimes very troublesome, particularly toward the termination of the disease. Black stools will be passed if there has been a large gastric hæmorrhage. There is sometimes present the albuminuria of anæmia and cachexia. Acetone and diacetic acid are sometimes found in the urine, and indican is often in excess. Peptonuria is occasionally seen.

A steadily advancing anæmia is one of the most constant symptoms of cancer. The number of red blood-cells is finally much reduced, as is the percentage of hæmoglobin. Usually they do not fall below 50 per cent. of the normal. Some degree of leucocytosis is the rule. Sometimes the intensity of the anæmia is greater, and approaches that of the pernicious type. In such cases poikilocytes, nucleated red blood-corpuscles, and numerous microcytes may be found. The condition differs, however, from pernicious anæmia both in the method of its production (it is not hæmolytic, but cytogenic, and there is no marked increase of iron in the liver) and in the fact that emaciation goes hand in hand with the anæmia. There finally develops the well-known cachexia commoner in this than in any other disease, although sometimes observed in other affections and not always present in cancer. The skin becomes harsh, has a faded yellow tint, and sometimes exhibits brownish spots, and the face is pinched and wrinkled. With this there is great loss of strength.

Dependent upon the anæmia are such symptoms as œdema of the ankles and general anasarca. Ascites may be the result of the condition of the blood or of a secondary cancerous peritonitis. Palpitation and dyspnœa and hæmic murmurs may also develop as the result of the anæmia. With increasing weakness the pulse naturally grows more feeble and rapid.

Exceptionally there are cases of cancer of the stomach in which for a considerable period there is neither anæmia, cachexia, nor emaciation. Thus, for instance, I have found pyloric cancer quite unexpectedly at the autopsy of a middle-aged man who had been attacked in apparently good health by acute fatal pneumonia.

The temperature in gastric cancer is generally unaffected, but in advanced cases may be continuously or occasionally elevated. There are sometimes chills followed by sweating. Toward the end of the disease the temperature may be subnormal. The intellect is as a rule unaffected throughout. Sometimes toward the close of life delirium may supervene, or a condition of coma may develop, which is attributed by v. Jaksch to diaceturia, and called by him "*coma diaceticum.*" It is identical with that observed in diabetes. In this condition a state of restlessness and excitement develops, which gradually changes into one of coma, often accompanied by dyspnœa. In other cases coma may be the result of the attendant anæmia, of uræmia, or of metastatic cancer of the brain.

Complications.—Many of the complications of cancer of the stomach depend upon the development of metastatic growths in other parts of the body. Others are produced independently of these. Some of them have already been referred to in discussing the symptoms and pathology of the disease. Of the remaining, jaundice is not infrequent. It may depend upon pressure upon the bile-ducts by growths in the liver, pancreas, or portal lymphatic glands, or may be produced in more ordinary ways, as by duodenitis or impacted gall-stones. These calculi are indeed found present in a notable proportion of cases. Enormous carcinomatous enlargement of the liver may attend the jaundice or may develop without it. Simple, suppurative, or cancerous pylethrombosis may occasionally occur. Peritonitis, perhaps with ascites, may result from the extension of the cancerous disease or from other causes. Pleurisy and pericarditis with effusion may likewise arise independently of, or dependent upon, secondary cancerous involvement. Pericarditis, however, is very unusual. Pyopneumothorax and pulmonary abscess or gangrene are occasional complications. Pulmonary œdema and pneumonia may develop in advanced cases as a result of the cachectic state, and aphthæ of the mouth, pharynx, and œsophagus may be produced under similar conditions. In the late stages marasmic venous thrombi, followed by painful local œdema, may form in the femoral or saphenous veins, or less often in the subclavian, axillary, or other veins. Emboli from these may enter the lungs. Tuberculosis sometimes complicates cancer. The frequency of chronic gastritis has been already mentioned. Dilatation of the stomach is often a direct result of the morbid growth, and diphtheritic inflammation of the colon is not uncommon in the later stages. Chronic nephritis is an occasional complication and in some cases explains the comatose phenomena above described.

Diagnosis.—The discovery of a tumor in the epigastrium, if combined with the other symptoms of cancer, renders the diagnosis certain. Without this tumor the diagnosis always possesses an element of doubt. Nevertheless, vomiting of coffee-ground material, progressive loss of flesh and strength, increasing anæmia, gastric pain and other symptoms of gastric disturbance, together with the constant absence of free hydrochloric acid from the gastric contents, render the diagnosis of carcinoma very probable even in the absence of any discoverable tumor. Probably the most important of these symptoms is the coffee-ground vomiting.

Cancer of the stomach must be differentiated from chronic gastritis, and particularly from gastric ulcer, with which it has many symptoms in common. The following table of diagnostic symptoms, taken from the article by Welch already referred to, illustrates the differences and likenesses so well that I have inserted it in full:

Gastric Cancer.	*Gastric Ulcer.*	*Chronic Catarrhal Gastritis.*
1. Tumor is present in three-fourths of the cases.	1. Tumor rare.	1. No tumor.
2. Rare under forty years of age.	2. May occur at any age after childhood. Over one-half of the cases under forty years of age.	2. May occur at any age.
3. Average duration about one year, rarely over two years.	3. Duration indefinite; may be for several years.	3. Duration indefinite.
4. Gastric hæmorrhage frequent, but rarely profuse; most common in the cachectic stage.	4. Gastric hæmorrhage less frequent than in cancer, but oftener profuse; not uncommon when the general health is but little impaired.	4. Gastric hæmorrhage rare.
5. Vomiting often has the peculiarities of that of dilatation of the stomach.	5. Vomiting rarely referable to dilatation of the stomach, and then only in a late stage of the disease.	5. Vomiting may or may not be present.
6. Free hydrochloric acid usually absent from the gastric contents in cancerous dilatation of the stomach.	6. Free hydrochloric acid usually present in the gastric contents.	6. Free hydrochloric acid may be present or absent.

7. Cancerous fragments may be found in the washings from the stomach or in the vomit (rare).	7. Absent.	7. Absent.
8. Secondary cancers may be recognized in the liver, the peritoneum, the lymphatic glands, and rarely in other parts of the body.	8. Absent.	8. Absent.
9. Loss of flesh and strength and development of cachexia usually more marked and more rapid than in ulcer or in gastritis, and less explicable by the gastric symptoms.	9. Cachectic appearance usually less marked and of later occurrence than in cancer, and more manifestly dependent upon the gastric disorders.	9. When uncomplicated, usually no appearance of cachexia.
10. Epigastric pain is often more continuous, less dependent upon taking food, less relieved by vomiting, and less localized than in ulcer.	10. Pain is often more paroxysmal, more influenced by taking food, oftener relieved by vomiting, and more sharply localized than in cancer.	10. The pain or distress induced by taking food is usually less severe than in cancer or in ulcer. Fixed point of tenderness usually absent.
11. Causation not known.	11. Causation not known.	11. Often referable to some known cause, such as abuse of alcohol, gormandizing, and certain diseases, as phthisis, Bright's disease, cirrhosis of the liver, etc.
12. No improvement or only temporary improvement in the course of the disease.	12. Sometimes a history of one or more previous similar attacks. The course may be irregular and intermittent. Usually marked improvement by regulation of diet.	12. May be a history of previous similar attacks. More amenable to regulation of diet than is cancer.

Nevertheless, in spite of these points of difference, the diagnosis is sometimes difficult. It must then rest more on the general complexus of symptoms than upon any one of them.

Certain other affections show more or less resemblance to gastric cancer. In its earlier stages cancer may simulate one of the neuroses of the stomach, but this resemblance will not continue long. It has been especially in cases where the cancer has involved the posterior wall and has formed adhesions posteriorly that recurring paroxysms of pain without discoverable tumor has simulated gastralgia for a considerable time. Stenosis of the pylorus, not the result of cancer, occurs usually at an early period of life, and is liable to have been preceded by evidences of ulcer. Its symptoms are likewise, as a rule, of longer duration. Nevertheless, it cannot in some cases be distinguished from the malignant stenosis. So also the induration which sometimes attends an old gastric ulcer, even though not at the pylorus, may be discoverable by palpation, and might arouse suspicions were it not that the history of the case is generally quite different from that of cancer. Cirrhosis of the stomach, the result of chronic gastritis, sometimes presents a tumor to palpation, but the affection is of long duration and is less liable to produce hæmorrhage and pain. It can often not be differentiated with any positiveness.

In cases where a tumor can be detected it is frequently difficult to determine whether this belongs to the stomach or to some other structure, especially the left lobe of the liver or the pancreas. Tumors of the liver are usually moved by respiration and are duller on percussion, while those of the stomach are generally more readily moved by the hand. When, however, a tumor of the stomach is adherent to the liver, the diagnosis will depend upon the symptoms in general. Cancer of the pancreas cannot be distinguished from cancer of the stomach by physical examination, and often not by any other means. It exhibits less pain after the ingestion of food and less tendency to cause dilatation of the stomach, hæmatemesis, anorexia, and vomiting. On the other hand, there may be jaundice, glycosuria, and fatty stools, and the pain is more frequently referred also to the back. Even the head of the normal pancreas may, in much emaciated individuals, give to the fingers the sensation of a tumor of the stomach.

A gastric cancer may receive an impulse transmitted from the aorta beneath it. There is lacking, however, the expansile quality of the pulsation of an aneurism. The general symptoms, too, are totally different.

Prognosis and Course.—The course of the disease has already been sufficiently described. It is always progressively from bad to worse, although often with occasional periods of temporary improvement in one or another symptom. In rare cases, considerable improvement in all the symptoms may occur and may even persist for some months, while the constant presence of the tumor forbids substantial encouragement. This may occur when an erroneous dietary and undue exertions are corrected ; but the more marked instances are seen when pyloric stenosis, which has caused extreme marasmus by interference with digestion, is removed by ulceration of the growth. I have known the improvement thus permitted to be prolonged and delusive. The duration of the disease can only be determined approximately and within rather broad limits. In early life its course is more rapid than in advanced years. Various estimates place the average duration at from twelve to eighteen months. In rare instances the duration is much longer and extends over several years. The prognosis is absolutely unfavorable as to the final result, and patients probably never recover. Operation might result in permanent recovery, but has not yet done so. Death takes place from exhaustion. The patients pass into a condition of collapse, often with the mind clear or, in some cases, with slight delirium, stupor, or coma. Perforation, hæmorrhage, and various complications are the causes of the fatal issue in a proportion of the cases.

Treatment.—Unfortunately, very little can be said under this heading. The treatment must be entirely palliative and symptomatic. Condurango was at one time vaunted as a specific. It has, however, no such property, although it is an excellent stomachic and in some cases relieves vomiting and gastric pain. It may be given in the form of a decoction, wine, or fluid extract.

In the line of symptomatic treatment is the relief of pain. For this purpose opium in some form is often required. No fear need be entertained of creating a habit, since life can in any case last but a short time. The size of the dose should, however, be restricted, lest the remedy cease to exert any beneficial action. Its constipating power is likewise a disadvantage. Sometimes chloroform or Hoffmann's anodyne internally, or counter-irritation externally, is efficient if the pain be not very severe.

Vomiting may be treated with bismuth alone or combined with morphine, chloroform, ice, carbonated water, champagne, hydrocyanic acid, or counter-irritation. In very obstinate cases it may be well to feed for a time by the rectum. Hæmatemesis, if severe enough to demand interference, is best treated by the swallowing of ice and by its application to the epigastrium, combined with the administration of ergot hypodermically.

Constipation may be treated by enemata, glycerin suppositories, or by mild laxatives. The patient should never be purged. Diarrhœa is best controlled by suppositories or enemata containing opium.

General symptoms of indigestion, such as acid and gaseous eructations, gaseous distention and the like, may be treated by antacids and antifermentatives, as soda, salicylic acid, and especially hydrochloric acid. The employment of the latter with or without pepsin is often indicated, since the digestive power of the gastric juice is so seriously interfered with in this disease. In a number of cases I have secured temporary benefit from the administration of carefully prepared emulsion of cod-liver oil with hypophosphites or with fluid extract of wild cherry. Where obstruction of the pylorus has caused dilatation of the stomach, and even in cases without dilatation, better results are frequently obtained by lavage of the stomach than by any other treatment.

The diet should be nutritious and very easy to digest. Starches can be better born than albuminoids, since there is a lack of digestive power in the gastric juice. They are, however, liable to undergo fermentative changes. It is often best to use such substances as previously digested albuminoids, peptonized milk, koumyss, and the like. When, however, the patients are able to take a somewhat selected mixed diet without discomfort there is not the need to restrict it greatly which is found in gastric ulcer.

The question of operative interference in cases of gastric cancer cannot yet be settled. The operations which have been performed for cancerous stenosis of the pylorus have not resulted very favorably. Many of the patients have died as a result of the operation, yet in a few instances life has been prolonged. In the light of the fact that purely medicinal treatment affords no promise whatever we must await with hope further experience in operative procedures.

NON-CANCEROUS TUMORS OF THE STOMACH.

These are rare and generally not productive of any symptoms. When symptoms are present and when the tumor is large enough to be felt, there is no way of determining that the growth is non-cancerous, excepting by observation of the course of the case. The commonest tumors are adenomatous polypi which project into the gastric cavity. They are composed of hypertrophic mucous membrane. The submucosa is not involved. They are of small size—about that of a bean—and often very numerous. They are supposed to be the result of chronic gastritis and are referred to in discussing that affection.

Adenomata of benign nature may rarely develop in the submucous tissue. Myomata and fibro-myomata in the form of polypi project either into the gastric or into the peritoneal cavity. They are sometimes of very large size. Sarcomata are rare. They may be either primary or secondary. They are said to have less tendency than cancer to ulcerate. A cystic myo-sarcoma weighing twelve pounds was described by Brodowsky. Secondary lympho-sarcomata have been reported. Lymphomata may occur in connection with leukæmia and pseudo-leukæmia. Lipomata and fibromata are very unusual. Dermoid cysts have been observed and other cystic growths have been described. Cases have been reported in which foreign bodies, particularly balls of hair, were present in the stomach and were supposed to be tumors.

Dilatation of the Stomach.

Definition.—An abnormally great and chronic enlargement of the stomach, with interference with the digesting and propelling power of its walls. When the stomach is simply unusually large, but unattended by characteristic symptoms, we have to do with *Megastria*, which is a condition, not a disease.

Synonyms.—Gastrectasis ; Gastrectasia.

Etiology.—Various causes operate alone or combined to produce the dilatation. They may be classified into two groups : (1) Mechanical obstruction of the pylorus ; (2) Insufficiency of the expelling power of the gastric walls. The latter may exist without the former. The former, however, is very liable eventually to produce a loss of muscular power ; although there is, in some cases, considerable stenosis without much dilatation, the muscular tone of the stomach being sufficient to overcome the obstruction.

(1) Narrowing of the pylorus or adjacent portion of the stomach or intestine is the cause of the severest cases of gastrectasis. The obstruction may be produced in various ways. Among the sources of obstruction situated in the stomach itself carcinoma heads the list as the most frequent, the next in importance being cicatricial contraction after simple gastric ulcer. Among other rarer causes are cicatrization after phlegmonous or toxic gastritis, congenital stenosis, simple hypertrophic stenosis not the result of cancer, and the obstruction of the pylorus by mucous polypi the result of gastritis. Both spasm of the pyloric muscle and a sharp bend or twist of the duodenum resulting from the weight of an overdistended stomach have been suggested as possible causes, but without positive proof that they occur.

Pressure from without may also narrow the pylorus or duodenum and produce gastric dilatation. Thus, there may be pressure by a tumor of the pancreas, liver, retroperitoneal glands, or omentum, or by a floating kidney or a gall-bladder filled with calculi. It is not probable, however, that such an easily movable body as a floating kidney could exert sufficient pressure to produce dilatation. Cicatricial bands resulting from former peritonitis may also cause dilatation by producing a stenosis, and a large scrotal hernia containing transverse colon or omentum has been known to produce stenosis and dilatation, probably the result of a sharp bend in the duodenum caused by the traction.

(2) Insufficiency of the expelling power of the stomach may exist and pro-

duce dilatation without the presence of any obstruction at the pylorus. The degree of dilatation is usually not as great as in cases due to stenosis. The insufficiency may be absolute or relative only. In the former the stomach has not the power to expel properly even food which is normal in quantity and quality. In relative insufficiency the muscular power is normal, but not great enough to manage gastric contents of abnormal quality or quantity.

The ingestion of excessive amounts of food and drink is a common cause of relative insufficiency. The condition is often seen in employees of breweries as a result of the very large amount of beer repeatedly taken by them. The enormous appetite of diabetics and of some cases of insanity acts in the same way.

Abnormalities in the quality of the food, developing before or after it is eaten, are likewise powerful factors. Chronic gastritis is consequently a common cause of the disease since, when it is present, the food is not properly digested, remains too long in the stomach, and overburdens it. The fermentative changes going on in the stomach with the production of gases tend to produce dilatation, since the irritation which these set up probably closes the sphincters and compels the contents to be retained. Both food and gas press upon the walls and stretch them. These fermentative changes may take place either when the hydrochloric acid is diminished or when it is normal, depending upon the number of micro-organisms introduced. On the other hand, hypersecretion of hydrochloric acid, as seen in some cases of nervous dyspepsia, may also favor dilatation, since it prevents the digestion of the starches which consequently tarry too long in the stomach.

Whether actual stenosis exist or whether there be relative insufficiency of the expulsive power of the stomach there will at last follow an absolute insufficiency of the muscular tone. This impairment of the muscular power of the stomach may, however, be produced independently. Among its causes are general muscular debility, as in chlorosis, anæmia, tuberculosis, neurasthenia, rickets, and severe illnesses of any kind. Chronic gastritis, too, actually weakens the muscular power by inflammatory changes, beside leading to abnormal changes in the food ingested. It is probably the most frequent cause of gastrectasia dependent upon muscular insufficiency. Chronic peritonitis, and, rarely, constipation sometimes seem to influence the motor power of the stomach in some reflex way. Cancer and ulcer, even though not at the pylorus, may impair the expelling power, and various degenerations, as the amyloid and colloid, may attack the muscles of the stomach and produce dilatation. It has been claimed, though not proven, that œdema of the gastric walls resulting from nephritis, cirrhosis of the liver, heart disease, or emphysema may cause dilatation. Finally, adhesions or herniæ may produce the disease by restraining the movements of the stomach instead of by occasioning stenosis, as before described.

Rarely, if ever, does but a single one of the causes mentioned operate alone. Rather do the various factors act and react upon each other, gradually and constantly increasing the gravity of the condition. For instance, stenosis is sure to be followed at last by muscular insufficiency, the result of the great retention of food. Fermentation, too, takes place in the food thus accumulated, and causes chronic gastritis, which in its turn both diminishes still further the muscular power and lessens the secretion of gastric juice, and each of these conditions increases the delay of the food in the stomach and the production of fermentative changes.

Although dilatation of the stomach may develop at any time of life, it is much commoner in those of middle age and older. It is however not infrequent in children, especially in those suffering from rickets.

Pathology.—The degree of dilatation of the stomach varies considerably. According to Ewald, the stomach can be said to be actually dilated only when it will contain more than from fifty-three to fifty-seven fluidounces. This is, however, only an average measurement; for a small individual or one with an unusually small stomach may develop dilatation without the stomach's surpassing in its capacity the figures given.

Instances of decided dilatation are at once recognized on post-mortem examination. On exposing the abdominal viscera the intestines are found forced into the pelvis, and the diaphragm, liver, and spleen pushed upward. The stomach may occupy most of the anterior portion of the abdomen. Its lower border in average cases reaches to between the umbilicus and the pubis. The fundus is generally the portion first and most dilated. This is especially true in the cases which result from stenosis, and it may occupy all of the left side of the abdomen in extreme cases. Unless adherent to surrounding parts the pylorus is very commonly somewhat depressed. Nevertheless, the general position of the stomach as a whole is more horizontal than in normal cases. Dilatation of the œsophagus is sometimes present. Dilatation of the pylorus, as well as of the stomach, must result if the obstruction be in the intestine.

The walls of the stomach may be thickened, normal, or thin. If the former, hypertrophic dilatation is spoken of; if of normal thickness or thin, atrophic dilatation is present. In the cases occurring in young persons and due to stenosis, hypertrophic dilatation is very apt to be the condition present, and is especially liable to occur in the region of the pylorus. The hypertrophy is usually situated in the muscular layer, and is consecutive to cancerous or cicatricial pyloric stenosis; but in some cases there is a hypertrophy, usually of all the coats of the stomach, which occurs at the pylorus and itself produces the stenosis. This latter condition was described by Lebert as "hypertrophic stenosis of the pylorus." In many of the cases the process has been called into action by the cicatrization of an ulcer, but in others it appears to be an entirely independent affection.

In aged persons dilatation of the stomach is rarely attended by hypertrophy of the gastric walls, and in cases due to muscular insufficiency hypertrophy, if present, rarely reaches the degree seen in stenotic cases.

In all cases there is a tendency for atrophy at least of the mucous layer to take place if the course has been prolonged. The mucous membrane in most cases of dilatation exhibits some degree of chronic gastritis.

Histologically, there is found a new growth of connective tissue, often with a small-celled infiltration which occupies the space between the muscle-bundles. There is also a cellular infiltration of the submucous layer. The mucous membrane exhibits in different parts the various histological stages of chronic gastritis.

Floating kidney has been found associated with gastrectasis in a number of cases, and some writers regard the dilatation as the cause of the renal displacement. It seems more probable, however, that there is no actual causal relation between them.

Symptomatology.—These vary much according to the severity of the case and the nature of the cause. The symptoms of the primary cause often mask those of a gastrectasis or may, at least, cause them to be overlooked. In nearly all cases there is an increase of dyspeptic symptoms, and sometimes these remain the only evidence of dilatation. The appetite varies within wide limits, but is usually diminished, although sometimes greatly increased. Thirst is generally very great; the tongue is coated; eructation of liquid and of gas is very common and the breath may be offensive; a sensation of distention and pressure after eating is common; there may be epigastric tenderness.

None of these symptoms are characteristic. Vomiting, however, is a most important diagnostic symptom and a very common one. It may be accomplished with the greatest ease, or may be very exhausting from the violent retching. At first it occurs often and soon after eating, but as the dilatation increases it is less frequent and does not take place until some hours after a meal. The amount of material vomited may be enormous, reaching eight or ten pints or even much more than this in some of the reported cases. Portions of undigested food eaten several days before are very commonly observed.

If the vomited material—which is always in a fermented state, as shown by the numerous bubbles of gas—be allowed to stand in a cylindrical glass vessel, it will separate into three layers, the upper brownish and frothy, a much thicker middle one of turbid yellow-brown or dark-gray fluid, and a lower and heterogenous layer largely composed of solid matter. The odor of the vomit is often offensive and the reaction usually acid. Microscopical examination shows a variety of moulds and bacteria, together with the yeast fungus and the sarcina ventriculi, with crystals of fatty acids and remnants of food of all sorts.

Hydrochloric acid may be absent from, or diminished in amount in the vomit in cases dependent upon carcinomatous stenosis, or it may be normal or increased in other cases. The great degree of fermentation leads to various products of decomposition, among them lactic, butyric, and acetic acids, alcohol, and various gases.

Vomiting is not always present in dilatation. So, too, the vomited matter does not always have the peculiar characteristics described.

In this connection may be mentioned the fact that although the secretory power of the stomach need not be affected in dilatation, and the character of its secretion not be at all altered, its digestive power is probably inhibited by the presence of unabsorbed peptones.

The iodide-of-potash test shows that the absorptive power of the stomach is seriously impaired, and the motor power of the organ is likewise in most cases greatly affected, as far as indicated by the salol test.

Although diarrhœa sometimes occurs, constipation is a symptom nearly always present, due in part to the fact that so small a quantity of food enters the bowel. The urine is scanty in well-marked cases with much vomiting. Its acidity is generally diminished, and sometimes the reaction is persistently alkaline. Acetone and diacetic acid may be present.

The nutrition of the patient varies considerably. In mild cases it may be little interfered with, but as the disease advances anæmia, weakness, and emaciation are prone to develop and may become extreme ; the skin is dry and roughened, and marantic œdema may appear. The spirits are depressed and headache, vertigo, and insomnia are common. Coma develops in rare cases, and it appears to depend upon diacetæmia. The temperature is generally unaffected. Palpitation and dyspnœa sometimes result from flatulent distention.

Kussmaul was the first to describe tetanoid spasms dependent upon gastric dilatation. The spasms were seen only in advanced cases, and developed after profuse vomiting or the employment of lavage. They sometimes began with painful sensations in the region of the stomach and with oppression and prostration. They were painful, tonic, and appeared in the flexors of the foot, arm, hand, and calf and in the abdominal muscles. Sometimes they were general. The pupils were contracted. The spasms lasted from a few minutes to a few days. Occasionally epileptiform convulsions with unconsciousness occurred.

Physical examination of the stomach reveals the most important symptoms. If the abdominal walls be thin and the stomach decidedly dilated, inspection usually reveals very evident bulging of the left hypochondriac, epigastric, a portion of the right hypochondriac, and a portion of the umbilical regions. The greatest prominence is on the left side, and, when the patient is standing, below the navel. The outline of the stomach can sometimes be distinctly seen. The greater curvature in such a case runs from the anterior end of the tenth rib on the left side toward the pubis, and then curves up again to the right costal margin, while the lesser curvature, not so often visible, may be seen a short distance below the ensiform cartilage. Artificial distention of the stomach in the manner already described elsewhere is useful in bringing into view the gastric boundaries if these were not previously visible. The procedure is not always successful, either because the pylorus allows the gas to pass out or because the stomach is so large that the effervescent powders do not produce enough gas to distend it.

In cases due to stenosis, and in which there is hypertrophy of the muscular wall, active peristaltic waves may occasionally be seen traveling over the stomach from left to right. Antiperistalsis is sometimes, although rarely, observed. Pressing or striking the abdomen with the hand will sometimes bring the movements into play in cases where they do not otherwise occur.

Palpation may detect a pyloric tumor in cases of dilatation from stenosis.

The hand perceives peristaltic movements if present, even although they cannot be seen, and also recognizes a peculiar elastic resistance which has been compared to that of an air-cushion. Leube recommended that a stiff sound be introduced in cases of suspected dilatation, and that the end of it be felt for through the abdominal walls. He stated that if this could be perceived below the umbilicus dilatation was present. The procedure is not to be recommended for the reasons explained in discussing the methods of examining the stomach.

Percussion can best be practised after the stomach has been artificially distended with air or gas. The limits of the organ can then be well outlined in many cases. Often, however, it is impossible to distinguish between the stomach and the distended transverse colon. It is therefore necessary to seek for the line of dulness caused by the presence of the solid and liquid gastric contents. In doing this, percussion should be performed when the patient is in the upright position. A line of dulness will be noticed in the dependent portion and marks the lower boundary of the stomach. This dulness, however, will shift or disappear entirely when the patient is recumbent. Dulness as low as or lower than the umbilicus indicates dilatation, provided there can be excluded the total displacement of the stomach downward, such as sometimes occurs in subjects with greatly relaxed abdomens. To bring out the dulness it is sometimes necessary to administer to the patient a considerable quantity of water by one of the methods for the examination of the stomach already described.

Under auscultation may be first mentioned the detection of the distinctly splashing sounds frequent in dilated stomachs. The patient being recumbent, the whole body may be rapidly shaken or the gastric region given a series of rather forcible and sudden impulses with the hand. In this way a loud splashing can often be heard with the stethoscope, or in other cases is even audible at a distance from the body. The simple presence of the sound is not diagnostic, since it may be heard in the healthy stomach immediately after a large amount of liquid has been swallowed or on the transverse colon. Dilatation, however, may be diagnosed if the sound can be elicited two or more hours after drinking, and if, also, siphoning out the stomach renders the sound no longer obtainable, and thus shows that it was actually produced in it and not in the colon. A fine, sizzling sound can often be distinguished by auscultating the stomach. It is accounted for by the fermentation in progress. It can also be heard when carbonic dioxide is being generated after effervescing powders have been given. The transmitted heart-sounds, with an added metallic quality, are sometimes very clearly audible over a dilated stomach filled with air or gas. It is often useful to practice auscultation by applying a stethoscope over the stomach while an assistant performs percussion at points advancing upwards from the lower part of the abdomen. The sudden and marked increase in the force and clearness of the transmitted sound may be noted when the true gastric area is reached. The development of the metallic percussion note, as employed in pneumo-thorax, may also occasionally be employed to outline the organ.

Direct mensuration of the stomach is unsatisfactory. A stiff sound passed into the stomach should normally enter about twenty-four inches from the incisor teeth, while in cases of dilation it may pass in as far as twenty-eight inches. The fallacies of this and other means of mensuration have already been referred to in discussing the methods of examining the stomach.

Diagnosis.—The diagnosis is usually easy if the symptoms detailed are borne in mind; but often, too, it is difficult or impossible. The most important features from this point of view are the physical signs of dilatation, the character of the matter vomited, and the information as to the delay in digestion obtained by the use of the stomach-tube. Of the results of the physical examination, the determination of the lower border of the stomach is the most important, for although the knowledge of this does not always indicate the degree of dilation, yet when this border is below the umbilicus, the diagnosis of dilatation can pretty safely be made. The important features of the vomited matter are the large amount of it, its occurrence some considerable time after a meal, its separation into three layers, and the evidence of fermentation. The most important diagnostic symptom learned from lavage is the evidence of delay in the digestion of food and of loss of the motor power of the stomach. This is shown by the presence of food in the stomach six to seven hours after a meal, by which time it ought to have entered the intestine.

Yet none of these symptoms are diagnostic in every case, and great care must be used lest they mislead by their occasional absence, or by their doubtful import when present. It is impossible to distinguish between the early stages of gastrectasis on the one hand, and between a naturally large stomach with chronic gastritis on the other. So, also, functional atony of the gastric muscle, existing as a pure neurosis, cannot be distinguished from the early stages of actual dilatation.

Prognosis and Course.—The prognosis depends largely upon the cause of the condition. In cases due to stenosis from malignant disease it is totally unfavorable, although there may be periods of temporary improvement, the result of proper treatment. Cases of dilatation from stenosis due to other causes offer a very favorable prognosis as regards duration of life, but permanent recovery is not to be expected, although careful treatment will make practicable a comparatively comfortable existence. In dilatation not dependent upon stenosis the prognosis is more favorable, although even here we can hardly expect to have the normal tone fully restored when once lost, and to see the patient's stomach return to the normal size if dilatation has been considerable and if the gastric walls have undergone serious histological changes. In the early stages of this class, where the dilatation is but moderate, permanent and complete recovery is to be hoped for. In cases which fail to respond to treatment death at last occurs, usually from a progressive marasmus.

Treatment.—The most important method of treatment of gastrectasia is lavage, which was introduced by Kussmaul in 1867. This is absolutely indispensable as a palliative measure in cases due to stenosis or in the severer instances of the non-stenotic class; and, indeed, in nearly every case of whatever degree of gravity it is necessary. By the use of lavage the burden of the irritating, fermenting gastric contents is removed, and the stomach is thoroughly cleansed and given an opportunity to recover its lost tone and to contract again. Lavage, systematically employed, gives remarkable results in the relief of the distressing symptoms. The sensation of distention, the annoying vomiting, and the evidences of dyspepsia in general, with many of the symptoms attending them, are removed by the treatment; while the stomach frequently diminishes in size with great rapidity, food can be readily taken and better digested and absorbed, and the nutrition of the patient often improves remarkably.

Lavage is best done by siphoning. A piece of flexible tubing, three or more feet in length and with a funnel attached, should be connected with an ordinary stomach-tube. The total length of the apparatus should be about six feet. Whatever contents are present in the stomach may be removed through this, and then about a pint of tepid water allowed to run in. The funnel is then depressed and the liquid siphoned out, and this process is repeated until the water comes away clear or nearly so. If on the introduction of the tube, the stomach is not roused to contraction so that on lowering the funnel the contents are discharged, it is necessary to pour in warm water until the stomach is fairly distended, when siphonage may readily be started. Sometimes a 2 per cent. solution of bicarbonate of sodium is preferable to simple warm water, on account of its solvent action upon the mucus. To check the fermentation some mild and harmless antiseptic solution, as one of salicylic acid of ½ per cent. strength, may be employed as the last washing of the operation.

Lavage should be done once a day or occasionally not so often as this. An hour as far removed as practicable from that of the ingestion of food should be selected, in order that the digestion and absorption of the meal may be interfered with as little as possible. It is well to perform the operation either before breakfast or half an hour before the midday meal. The patient can usually learn after a few trials to wash out his own stomach.

There are certain objections to the employment of lavage. Sometimes the operation is so distressing to the patient that it has to be abandoned. It is questionable whether the tube should be inserted in cases of gastric ulcer, and recent hæmorrhage or the presence of aortic aneurism is an absolute contraindication.

Equally important with lavage is the careful regulation of the diet. Food should be of the most digestible nature and taken in small quantities only, and should consist of articles which do not readily ferment. It is very necessary that as little liquid as possible be ingested, since this remains in the stomach without being absorbed. Alcohol is to be avoided unless really indicated, and so, also, are starches, sugar, and fat in more than small amounts. Peptonized meat preparations are often of great value. Nutritive enemata or peptone suppositories are useful in some cases.

Measures must also be taken to increase the muscular power of the stomach as far as possible. For this purpose strychnine is indicated, and much may be expected from it in cases not too far advanced. It is best given hypodermically in cautiously increasing doses. Faradization of the stomach may increase its muscular power and hasten the passage of its contents into the intestine. The effects of massage are similar, although it acts principally by mechanically forcing the food through the pylorus.

In suitable cases fermentation can sometimes be checked and digestion aided by the administration of hydrochloric acid. Other remedies, as creasote, carbolic acid, naphthalin, salicylic acid, thymol, and allied substances are also at times of value. Carlsbad water has been advocated to relieve the constipation which so usually attends and reflexly to increase the peristalsis of the stomach.

Forcible dilatation of the pylorus narrowed by cicatricial contraction has been recommended by Loretta, and a number of cases have been very greatly benefited by it, although the operation is a serious one and the mortality high. Resection of the pylorus has been done in this and in cancerous stenosis. Other operative procedures have been proposed and performed, but their consideration belongs rather to works upon surgical subjects.

ACUTE DILATATION OF THE STOMACH.

This is a rare condition, of which our knowledge is largely theoretical and of which only a few cases have been described. The rapid ingestion of enormous quantities of liquid or solid substance, injuries of the abdomen, and convalescence from acute fevers are assigned as causes. The evidence of extreme paralytic dilatation comes on rapidly. The condition has appeared in some cases to be, so to speak, an exacerbation of a chronic dilatation which has previously given few, if any, evidences of its existence. The symptoms are severe pain in the abdomen, the absence of vomiting or its cessation if previously present, and great tympanitic distention of the stomach. The prognosis is grave and depends upon the nature of the primary disease.

Treatment consists in the evacuation of the contents of the stomach with a tube, with such subsequent measures as are called for by the causal conditions.

HÆMORRHAGE FROM THE STOMACH.

Etiology.—Hæmorrhage from the stomach (hæmatemesis; gastrorrhagia) is a symptom of so much importance, and one which may be produced in so many and so diverse ways, that a separate review of the subject is desirable. Among the causes to be mentioned are the following:

(1) *Traumata of Various kinds, Mechanical, Chemical, or Thermal.*—Blows upon the region of the stomach, penetrating wounds of the viscus, rarely the use of a stiff stomach-tube or of the stomach-pump, sharp-pointed foreign bodies which have been swallowed, are instances of mechanical causes of

trauma. Hot liquids and corrosive poisons, whether acid or alkaline, or of other nature, form thermal and chemical causes.

(2) *Ulceration of the Gastric Wall.*—In this category belong cancer, the peptic, tubercular and typhoid ulcers, hæmorrhagic erosions, phlegmonous gastritis and the penetration of an abscess from without through the wall of the stomach. By far the commonest causes among these are ulcer and cancer, the former tending to produce profuse hæmorrhage, and the latter repeated smaller ones. Ulcer may also in rare cases cause hæmorrhage by eating into the heart or the large vessels.

(3) *Diseases of the Vessels of the Wall of the Stomach.*—Fatty, amyloid, and atheromatous changes of the gastric vessels and also a varicose condition of the veins are to be mentioned here. Miliary aneurisms have been the cause of fatal hæmorrhage in some instances.

(4) *Active Congestion.*—Hæmorrhage is sometimes, though very rarely, seen in severe, simple acute gastritis. Vicarious menstruation by the way of the stomach is to be included here, as are also the reported cases of hæmatemesis substituting a hæmorrhoidal flux. Both of these are certainly very unusual.

(5) *Passive Congestion.*—A very frequent cause of this venous stasis and of hæmorrhage from the stomach is cirrhosis of the liver. Next to ulcer and cancer, it ranks as the most frequent causal condition. In a similar manner venous obstruction may follow pylephlebitis, pressure of a tumor upon the portal vein or upon its branches within the liver, pressure of distended bile-capillaries upon the smaller blood-vessels within the liver, and pigment-emboli resulting from melanæmia. Chronic diseases of the heart and lungs produce passive congestion of the stomach secondarily. Violent attempts at vomiting and the expulsive efforts of parturition may produce gastric hæmorrhage in the same way. Passive congestion is the cause in many instances of the varicose condition of the gastric veins already referred to.

(6) *Acute Infectious Diseases.*—The manner in which these produce hæmorrhage is not understood. Among those in which it occurs with more or less frequency may be mentioned yellow fever, small-pox, measles, scarlatina, relapsing fever, malaria, typhus fever, typhoid fever, cholera, diphtheria, and erysipelas. Hæmorrhage in most of these diseases is not common, and only occurs when there is evidence of the dissolution of blood in the system. The hæmatemesis of acute yellow atrophy may be included here, as perhaps also can that of acute fatty degeneration of the newly-born, described by Buhl.

(7) *Other Constitutional Diseases and Diseases of the Blood.*—Here are to be included hæmophilia, purpura, scurvy, chlorosis, pernicious anæmia, leukæmia, splenic anæmia, lymphatic anæmia, cholæmia.

(8) *Nervous Affections.*—Hysteria, progressive paralysis of the insane, epilepsy, and tubercular meningitis may be attended by hæmorrhage in some cases.

(9) *Idiopathic Hæmorrhages.*—Here may be included the cases described by Flint in which hæmorrhage of the stomach occurs just as it does from the nose, without any discoverable cause and neither followed nor preceded by any morbid condition.

(10) *Blood from Without the Stomach.*—Sometimes the blood does not come originally from the stomach, (gastrorrhagia), but enters it from without, and is then vomited. Thus it may come from the nose, lungs or pharynx and have been swallowed, or may have entered the stomach from a duodenal ulcer, a typhoid ulcer high in the small intestine or from the œsophagus, as in the case of rupture of varicose veins in the latter. The frequency with which, particularly in children, the blood, in cases of post-nasal hæmorrhage, flows down the œsophagus and is subsequently vomited renders this source of hæmatemesis especially worthy of note. In the case of children it may have come originally from the mother's breast in the process of nursing. The entrance of blood from the heart through a perforating gastric ulcer, already alluded to, and the bursting of an aneurism into the stomach are properly included here.

(11) *Melæna Neonatorum.*—In this disease blood may be vomited as well as passed from the bowel. It occupies an uncertain position among the forms of hæmorrhage from the stomach. Some of the cases depend upon ulcer and others on hæmophilia, but the nature of still others is uncertain.

Pathology.—This is most varied, depending upon the nature of the cause. The search for the source of the hæmorrhage is often difficult. In ulcer and cancer the lesion is easily found, but in hæmorrhage from miliary aneurism it readily escapes notice. In the case of hæmorrhage due to portal obstruction the source of the bleeding cannot usually be discovered.

Symptomatology.—Cases of slight hæmorrhage may readily present no symptoms, since the blood is entirely digested, and is therefore neither vomited nor passed in the stools. In instances of more abundant bleeding there are black passages, either hard or soft, and often of a very offensive odor. This has been sufficiently described in considering Ulcer and Cancer. Sometimes the stomach is so full of blood that a veritable cast of the organ is produced which may weigh several pounds.

With profuse gastric hæmorrhage there are, of course, the symptoms of sudden profound anæmia, and the patient may even die before any blood appears either in the vomit or in the stool.

Diagnosis.—Not only is the source of the blood to be determined, but also the cause of the hæmorrhage whenever possible. In cases of hæmatemesis it is, as a rule, easy to determine by chemical and microscopical examination that the colored material is really blood. That the blood is actually vomited is also generally clearly apparent if the patient has been seen in an attack or soon afterward. In doubtful cases hæmoptysis and hæmatemesis are to be distinguished as follows:

In hæmatemesis there are usually symptoms of gastric or hepatic disorder or of some of the other causes already mentioned; whereas in hæmoptysis there are generally evidences of actual pulmonary disease or of severe passive congestion of the lung from heart disease.

In hæmatemesis the blood is evidently vomited and there is no cough, except as a secondary symptom. The blood is bright red if vomited immediately after entering the stomach; otherwise it is clotted and mixed with portions of food, and is often of acid reaction. In hæmoptysis, on the other hand, the blood is coughed up. It is bright red, alkaline, frothy, little if at all clotted, and often mixed with mucus or pus. There is no vomiting, or it immediately follows the coughing.

In hæmatemesis the attack may be preceded by a sensation of fulness in the stomach, nausea, and perhaps faintness and dizziness; whereas in hæmoptysis there generally occurs a sensation of tickling in the throat before the blood starts.

After an attack of hæmatemesis there will probably be evidences of blood in the passages from the bowels; whereas in hæmoptysis cough and slightly blood-streaked expectoration persist after the attack is over.

The diagnosis of the exact cause of a gastric hæmorrhage is more difficult. Whether blood in the stools has its origin in the stomach or in the intestine can only be determined by the attendant symptoms, and not always then. The respective characters of blood vomited in ulcer and in cancer have already been described. Inspection of the nose and throat and, in the case of infants, of the mother's breast will often determine whether the blood has been swallowed and then vomited. Sometimes hysterical subjects or malingerers swallow the blood of an animal and then vomit it under the influence of emetics. Careful inquiry into the nature of the case generally reveals the deception.

Profuse hæmorrhage may occur in ulcer of the stomach, cirrhosis of the liver, pernicious anæmia, splenic anæmia, leukæmia, and aneurism. In these conditions, as in hæmorrhage from any other of the causes mentioned, only the general clinical history and concomitant symptoms can aid in the diagnosis.

Prognosis.—Hæmorrhage from the stomach is rarely immediately fatal, except in cases of aneurism. Ulcer, as already stated, may occasionally prove fatal by hæmorrhage, and hepatic cirrhosis does so with relative frequency. The symptoms of gastric ulcer and of cirrhosis of the liver are sometimes temporarily relieved by a hæmorrhage, yet the state of the patient can hardly be said to be improved.

CIRRHOSIS OF THE STOMACH.

Cirrhosis of the stomach (Hypertrophy of the gastric walls; Chronic interstitial gastritis; Fibroid induration of the stomach) has already been described as one of the later stages of chronic gastritis. It would seem occasionally to arise independently of this. It is characterized by very great increase in thickness of all or part of the gastric wall. It is a rare disease, commoner in women than in men, and occurring usually in middle age.

The causes are not clearly understood. Excessive use of alcohol is frequently assigned as one of them, but it is not certain with how great reason; since in some instances reported there had been no use of alcohol whatever. The cicatrization of a gastric ulcer has been supposed to be the starting-point of the disease in some instances. Trauma received in the region of the epigastrium has also been assigned as a cause.

The stomach is sometimes very greatly contracted, and holds but a few ounces of liquid. Such excessive contraction, however, is not usual, and the organ may be normal in size or even dilated. In the latter case the cirrhosis has been only partial. The walls of the stomach are greatly thickened and are rigid when incised. The thickness may measure even an inch or more. Microscopically, there is found a very great overgrowth of connective tissue in the submucous layer, great hypertrophy of the muscular coat, and hypertrophy also of the muscularis mucosæ. The mucous membrane may be normal, but is oftener more or less atrophied. In the case reported by Marcy and Griffith there was a new formation of smooth muscular fibre not only in the muscular layer, but in the submucous layer as well.

The symptoms are not at all characteristic, and sometimes there are none. In general there are the evidences of chronic indigestion, such as vomiting, oppression, loss of appetite, and the like. Gastralgia is sometimes marked. In other instances the symptoms of pernicious anæmia have been reported, and quite frequently the disease has simulated cancer of the stomach. A tumor may occasionally be detected in the epigastrium. The introduction of the stomach-tube and the observation of the short distance to which it enters, and the results of an effort to fill the stomach with water, may sometimes aid in the diagnosis where the organ is much contracted. It is, however, almost impossible to distinguish the disease from cancer in many cases.

The prognosis is entirely unfavorable, and the treatment is symptomatic.

HYPERTROPHIC STENOSIS OF THE PYLORUS.

Hypertrophic stenosis of the pylorus has been referred to among the causes of dilatation. It is usually the result of cancer, but cases have been described (notably by Lebert) in which it seems to arise as an independent affection. In such instances either one or all of the layers of the gastric wall in the neighborhood of the pylorus are hypertrophied. Where the whole thickness of the wall is involved the case is practically one of localized cirrhosis of the stomach.

The symptoms, prognosis, and treatment have already been discussed in considering Dilatation of the Stomach.

ATROPHY OF THE GASTRIC MUCOUS MEMBRANE.

Atrophy of the gastric mucous membrane has already been alluded to as the final stage of chronic gastritis. It may also be secondary to cancer and cirrhosis of the stomach. A number of writers, however, consider that it may arise entirely independently of these, and that it then constitutes a distinct affection. The macroscopic and microscopic appearances have been already sufficiently described under Chronic Gastritis.

A close relation sometimes exists between atrophy of the gastric mucous membrane and pernicious anæmia, and cases have been reported in which this organic change was attended by all the symptoms of pernicious anæmia, with the addition of the evidences of entire inability of the stomach to digest the food which entered it. Upon this combination of profound anæmia and digestive disturbance the diagnosis of such cases may be made. Often, however, the digestive disturbances are not so well marked and a diagnosis is impossible.

The prognosis is absolutely unfavorable, and the treatment should be that which is recommended for the later stages of chronic gastritis.

GASTROMALACIA.

This is a subject which formerly received much greater attention than it deserved and than is now allotted to it. It is at present generally admitted that nearly every, if not every, case of gastromalacia consists simply of post-mortem digestion of portions of the gastric mucous membrane, or at least of digestion of portions which before death had sustained some severe anatomical lesion, such, for example, as extensive hæmorrhagic infiltration.

RUPTURE OF THE STOMACH.

Rupture of a previously healthy stomach may occur as the result of a severe injury to the abdomen. It has also been claimed, but not proved, that a healthy stomach may rupture from over-distention with food or gas, but most of such cases reported are probably instances of perforation of an ulcer. Rupture secondary to other lesions has already been repeatedly referred to in various preceding sections.

DISEASES OF THE INTESTINES.

BY WILLIAM PEPPER.

INASMUCH as many of the affections of various parts of the intestine are much alike in their causes and symptoms and since, as a rule, it is but seldom that only one section of the enteric tract is involved at a time, but rather that more or less of the whole tube is affected, it seems better to avoid a too narrow subdivision into diseases of the duodenum, jejunum, ileum, and so-forth, and to consider, as far as possible, the disorders of the tube as a whole.

ACUTE CATARRHAL ENTERITIS.

Definition.—An acute catarrhal inflammation of more or less of the large and small intestine.

SYNONYMS.—Acute diarrhœa; Acute intestinal catarrh; Acute ileo-colitis.

Etiology.—The affection is an exceedingly frequent one, and varies greatly both in its cause and in its intensity. It may arise either primarily or secondarily in the course of other affections. Among predisposing causes of the primary form, the period of the year occupies a prominent place, the affection being far more frequent in the hot season. The disease is far more common in children, and especially in those two years old and younger. Previous attacks render the subject liable to succeeding ones. There is also often shown a peculiar and very marked individual susceptibility to diarrhœa in persons otherwise entirely well. Insufficient clothing, sedentary or confining occupation, and debility of constitution likewise act as predisposing causes.

Among the exciting causes, by far the most frequent are atmospheric influences and the ingestion of food which is improper either in quantity or quality. A sudden fall in temperature, if the body be not properly protected; exposure to drafts; chilling of the surface after profuse perspiration, often induce diarrhœa. In regard to food, the amount may be too great and may act as an offending body and excite the inflammation. In other cases certain articles of diet produce the affection, although these vary with different individuals. Often poisonous decomposition-changes which have taken place in the food before ingestion act as the exciting cause, producing in this way a toxic enteritis.

Impurities in drinking-water may give rise to epidemics of enteritis. Very often a change in the character of the drinking-water, as experienced by travellers, induces a diarrhœa, although the water, as a rule, may be harmless to those accustomed to it. Unripe fruit is another cause of epidemics of diarrhœa.

Organic toxic substances other than those of decomposed food, and such inorganic poisons as arsenic, acids and alkalies, mercury, antimony and drastic drugs, may produce diarrhœa. The inhalation of air rendered impure by emanations from sewers or from dissecting-rooms appears often to induce it.

Trauma may rarely occasion enteritis, as, for example, by blows on the abdomen, foreign bodies, intestinal concretions, intestinal worms, and the irritation of hardened fæcal masses in prolonged constipation.

Intestinal catarrh sometimes occurs epidemically, preceding outbreaks of cholera or dysentery. Alterations in the composition of the intestinal secretions may produce enteritis. Thus, the absence of the pancreatic juice is often attended by a fatty diarrhœa, and the increase or decrease of the amount of bile may be followed by a similar result.

Nervous influences may produce diarrhœa, but this will be referred to separately, as it is hardly a catarrhal process.

Secondary catarrhal enteritis may develop as the result of the extension of inflammation from the stomach, from a cancerous or tubercular ulceration of the intestine, from an invagination, hernia, or peritonitis, or in the case of inflammation of the lower bowel as an extension from gonorrhœal vaginitis or eczema of the anus. It may also occur in the course of infectious diseases, as measles, scarlet fever, tuberculosis, small-pox, typhoid fever, cholera, dysentery, pneumonia, pyæmia, and malaria. It may attend also chronic cachectic diseases in general, as cancer, phthisis, nephritis, anæmia, and Addison's disease. Circulatory disturbances, especially those involving the portal circulation, are active causes of secondary enteritis. Such may arise as the result of disease of the heart or lungs, cirrhosis of the liver, disease of the portal vein, or pressure on this or the mesenteric veins by tumors.

Extensive burns of the skin may produce a secondary catarrhal enteritis in a manner not understood.

Pathology.—In many instances in which there have been very marked symptoms during life post-mortem examination shows none of the appearances which are generally assumed as characteristic of catarrhal inflammation—viz. swelling, redness, and increased secretion. In such cases the characteristic pathological appearances were probably present during life, but disappeared when death occurred.

If the anatomical changes are well marked, there is found decided redness of the mucous membrane, which is most profuse at the tips of the villi and on the folds of the mucous membrane. It is sometimes diffusely and uniformly spread, and sometimes occurs in patches or streaks or is arborescent. Hæmorrhagic spots are visible; oftener, however, the mucous membrane is rather pale than injected and is covered with mucus. There are also present decided swelling and softening of the mucous membrane, and usually, to some degree, of the submucosa as well. This swelling is the result of an inflammatory œdema rather than of a cellular infiltration, although there is some increase of connective-tissue cells. The solitary and agminated glands are enlarged and more prominent than normal, appearing often as gray translucent granulations surrounded by an areola of congested blood-vessels. Sometimes small erosions are seen in the centre of the follicles, and rarely more extensive ulceration in the surrounding mucous membrane may accompany the catarrhal process.

The muscular and serous layers of the intestinal wall are generally uninvolved, although sometimes here and there hyperæmic. The mesenteric glands are nearly always enlarged and hyperæmic.

Symptomatology.—The symptoms not only vary greatly in intensity, but necessarily differ according to the portion of the enteric tract chiefly involved. For example, the evidences of duodenitis and of typhlitis are widely different. The symptoms here described apply particularly to the commonest form of enteritis—viz. ileo-colitis.

Diarrhœa is nearly always the most characteristic symptom. The stools are watery, or sometimes like thin mush, and vary in number from two or three up to twenty or many more in twenty-four hours. The amount of material passed often appears to be out of all proportion to the food ingested. This is a proof that there is not only a hurrying along of the intestinal contents by increased peristalsis, but that a large amount of fluid is actually added to them by transudation from the inflamed mucous membrane. The appearance of the passages varies greatly. The color ranges from brown to yellow, green, black, gray, and nearly white, its depth depending upon the amount of bile present. Considerable mucus, often tinged with bile, may be visible, or small quantities of blood may be present. Portions of undigested food may be passed. Sometimes the stools are foamy and very ill-smelling, and in other cases nearly destitute of fæcal odor. In very severe cases they may much resemble the serous, rice-water discharges of cholera.

The reaction of the passages is generally alkaline. Microscopically, there are found epithelial cells, round cells, occasional red blood-corpuscles, innumerable bacteria, Charcot's crystals, and crystals of oxalate of lime, neutral phosphate of lime, ammonio-magnesium phosphate, cholesterin and the fatty acids.

Sometimes diarrhœa is the only symptom of enteritis. As a rule, however, other evidences of the disease appear. Abdominal pain of a colicky nature is generally present. It is often worse just before an evacuation and is frequently relieved by firm pressure upon the abdomen. A tendency to straining indicates decided involvement of the colon. The abdomen is often tympanitic, and sometimes greatly so, and there may arise therefrom decided interference with respiration. Palpitation of the heart may be produced in the same way. Borborygmi are frequent as the result of the rapid motion of the mingled liquid and gas, and the peristaltic movements of the intestine may be visible or can be palpated through the abdominal walls. Appetite is impaired or lost, but sometimes entirely preserved, the tongue is coated and somewhat dry and thirst is nearly always increased. The temperature may be elevated somewhat in proportion to the severity of the intestinal lesions, but, as a rule, it is inconsiderably so. High continued fever or sudden elevations of temperature in the course of the disease indicate the presence of some complication. The urine is much diminished in quantity and is high-colored and full of urates, and tube-casts are occasionally seen in it. Delirium may occur, especially in children.

Symptoms of collapse may develop when the attack has been very severe. This is especially true of children and of aged persons or when the abdominal pain is very intense.

As already stated, the symptoms described are those most commonly observed, and are characteristic of the majority of attacks of enteritis. In case of the localization of the inflammation in other parts of the intestine other symptoms may appear. Duodenitis is usually associated with gastritis. The occurrence of jaundice combined with gastric symptoms points toward its existence, as do dull pain and tenderness in the right hypochondrium. A positive diagnosis, however, can hardly be made.

Jejunitis and ileitis almost always exist together. They cannot be distinguished from each other during life. Although they are nearly always combined with inflammation of the colon, yet they may occasionally exist alone. In such cases diarrhœa is usually less, and may not occur at all, since, in the absence of increased peristalsis of the colon, there will probably be time enough for resorption of the excess of fluid to take place. There will, however, be colicky pain in the abdomen, while the presence of undigested food and of unaltered bile in the stools is a further indication of involvement of the small intestine.

Acute catarrhal colitis, occurring alone, is unusual, since the ileum is nearly always likewise involved. It cannot be distinguished with certainty from ileo-colitis. Pain is often entirely absent, or, if present, may be very intense, but it is not so colicky. Mucus is more abundant, and the stools are uniform in consistence and without undigested food.

Inflammation limited to the cæcum or appendix is not uncommon. The symptoms differ from those described, and will be detailed in a separate section.

Catarrhal proctitis exhibits very frequent stools with a constant desire to evacuate the bowels, and this is accompanied by straining, and preceded or attended by pain in the left iliac region. There is tenderness on pressure in this locality, and a sensation of burning in the rectum. There is also a large amount of mucus in the stools, and sometimes more or less blood. Direct examination commonly shows spasmodic twitching and contraction of the anus, and pain on the introduction of the finger into the rectum. After the disease has existed for a short time, a degree of paralysis of the sphincter develops, and fluid is constantly discharged in small amount. Prolapse of the rectum is liable to occur.

Complications.—The symptoms of acute gastritis are very frequently combined with those of acute enteritis. The various diseases to which enteritis is often secondary may be considered as complications as well.

Diagnosis.—The diagnosis of the usual form of enteritis—viz. ileo-colitis—is generally easy. If fever persists for several days, the suspicion of the presence of typhoid fever arises; and the discovery of rose-colored spots, with the presence of other characteristic symptoms, renders the diagnosis of the latter affection certain.

In times of the prevalence of cholera it may be impossible to distinguish between the milder cases of this disease and those of severe catarrhal enteritis. In such instances there should be made a bacteriological study of the stools in the search for the comma bacillus.

Catarrhal ileo-colitis is distinguished from dysentery by the presence of large watery stools and the absence of any considerable degree of tenesmus. Peritonitis has abdominal pain and tenderness, but these are much greater than in intestinal catarrh, and the disease is attended by constipation. Enteralgia exhibits constipation and no evidence of disturbance of digestion, while there are other nervous symptoms present. In rheumatism of the abdominal muscles the pain is superficial and made worse by pressure and movements of the body.

Prognosis.—The prognosis is almost always good, except in the most severe cases and in the very young or very old or in those already much debilitated by other diseases. There is also the danger of the disease becoming chronic.

The attack lasts one or two days, or may occasionally persist for a week with varying intensity and with intermissions.

Treatment.—This must depend largely upon the individual case. The patient should be confined to bed in the horizontal position and be fed only with nourishment of the blandest sort, such as boiled milk, milk and rice, or peptonized preparations; or, better still, all food should be entirely withheld for twenty-four hours. Only moderate amounts of water should be taken, and ice is, in fact, preferable for the quenching of thirst.

If the patient is seen early in the attack and there is reason to believe that irritating food is still present in the intestine, it is well to begin treatment with a purgative consisting either of castor oil, Rochelle salts, divided doses of calomel and soda or a dose of the following:

℞. Chlorodyni,	gtt. cx;
Syr. rhei aromatici,	f℥ij.—M.

Sig. A teaspoonful in water, repeated as required.

If, however, purging has been profuse or the disease has already lasted a couple of days, this treatment is not advisable. This does not always mean that the diarrhœa is to be quickly checked, since it will generally cease of itself.

If pain be severe, mustard plasters or turpentine stupes may be employed, or spirits of chloroform or some such aromatic as ginger be given internally. Often opium is required, and sometimes even morphine hypodermically.

If the diarrhœa be profuse or tend to persist, astringents must be employed. One of the best of these is bismuth, in doses of 6 to 10 grains every two or three hours. Each dose may be well combined with 2 grains of Dover's powder. Dilute sulphuric acid in doses of 10 drops may be administered with morphine, $\frac{1}{16}$ to $\frac{1}{8}$ of a grain, every two or three hours. With each dose of this combination 5 grains of gallic acid may be advantageously given. Chalk mixture is a very useful mild astringent, especially in young subjects. Among other remedies may be mentioned acetate of lead, nitrate of silver, and various drugs containing tannic acid.

Enemata or suppositories are often of benefit, especially in cases where the disease appears to be situated chiefly in the large intestine. Simple flushing of the bowel with warm-water in quantities of several pints, with or without small amounts of a simple disinfectant or sedative, is often of the greatest service. Small soothing or slightly astringent enemata or suppositories can be used where the lower bowel is the part chiefly or solely involved. For this purpose, a couple of ounces of starch water with a few drops of deodorized laudanum may be introduced and retained; or one ounce of cold water containing one grain of zinc sulphate or silver nitrate and 5 to 8 drops of deodorized laudanum may be used as an enema from one to three times daily.

In addition to the treatment described it is of course of the greatest importance to remove the cause of the disease and to prevent a return of its action after the attack is over, as also to treat any other disorder to which the enteritis may be secondary. As the patient recovers the greatest care must be exercised in the choice of the food.

The treatment of duodenitis consists principally in the regulation of the diet. Thus starch and fats should be withheld, inasmuch as the entrance of bile and pancreatic juice into the duodenum is interfered with. Counter-irritation over the right hypochondrium is useful.

The treatment of proctitis should be chiefly local, and consist of soothing injections or suppositories or the insertion of small pieces of ice.

CHOLERA NOSTRAS.

Definition.—An acute and excessively severe gastro-intestinal catarrh, presenting symptoms very similar to those of cholera.

SYNONYMS.—Cholera morbus; Sporadic cholera.

Etiology.—The disease is in reality the most intense form of acute catarrhal enteritis combined with severe acute gastritis. It is not certain, however, that it is only this, for in many respects the affection is peculiar.

It is most common in late summer under the influence of intensely hot days combined with decidedly cooler nights. The disease is most frequent in youth, and nervous exhaustion of any kind is also a predisposing factor.

Among directly exciting causes are to be mentioned taking cold, errors in diet, and the ingestion of impure drinking-water, decomposed flesh, or unripe fruit. It is very probable that chemical products from decomposition of food in the intestine are the cause of most attacks.

Pathology.—The pathological lesions are those of acute gastro-enteritis, and a repetition of the description of these is unnecessary. Often the lesions do not seem severe enough to account for the intensity of the symptoms.

Symptomatology.—The attack is usually very sudden, though sometimes preceded by malaise, nausea, and loss of appetite. The first marked symptoms are very liable to come on during the night. The patient is awakened from sleep suffering from a sense of pressure in the abdomen, nausea, and repeated vomiting. At first it is the ordinary gastric contents which are expelled, colored variously and often mixed with bile, but later the ejecta are almost entirely watery. Vomiting may be easily accomplished or may be very violent. It may be almost incessant. Purging develops simultaneously or very shortly. The stools are at first thin, but fæcal and of varying color. Later they may become almost odorless, and sometimes entirely colorless, and possess the character of the rice-water discharges of cholera. Diarrhœa may be very copious, and the evacuation of the bowels become almost incessant.

Pain in the abdomen is present, and is usually intense. It is paroxysmal, and comes on after each attack of vomiting or purging. Cramp-like pains with involuntary twitching develop in the muscles, and particularly in those of the calves.

Thirst is intense, and there is much restlessness. The urine is greatly diminished in amount, and albumin and casts frequently appear in it, and indican is in great excess. Sometimes there is entire anuria. Collapse is very liable to develop. It is marked by cold, clammy, cyanosed skin; sinking in of the eyes; sharpness of the features and particularly of the nose; weakening and alteration of the voice, which sometimes becomes peculiarly high-pitched; small, rapid pulse; cold breath; sighing respiration, and low temperature of the skin, although perhaps with elevation of the rectal temperature. Fever is, however, generally absent. The mind is frequently clear throughout, but lethargy and, later, stupor may arise.

Diagnosis.—This is generally easy except in seasons when Asiatic cholera is prevailing. The fact that the stools generally still retain some color is a point of difference between the two diseases. Nevertheless, in very many cases the stools of cholera nostras are identical in appearance with those of the Asiatic disease. In such cases a bacteriological study of the passages may aid the diagnosis, although the bacilli of the two diseases are very similar in appearance.

It must likewise be borne in mind that poisoning by antimony, arsenic, or some fungi may produce symptoms identical with those of cholera nostras.

Prognosis and Course.—The disease does not often last longer than from twenty-four to forty-eight hours, and in favorable cases the symptoms of collapse then disappear and vomiting and purging cease, perhaps leaving the patient weak for some days or even weeks. This is the event in the majority of cases, and nearly always so in previously healthy individuals. In the very old or very young or in those already debilitated death may take place. In fatal cases the diarrhœa and vomiting may cease entirely, and incessant hiccoughing often develops; or in the other instances the alvine discharges become almost continuous and involuntary and the cramps are nearly constant. Collapse increases, and coma may precede death.

Treatment.—The principal treatment consists in the prompt and free exhibition of opium, either by the mouth or, still better, hypodermically. Chlorodyne often acts admirably. Measures must also be taken to avert or remove symptoms of collapse. For this purpose the application of large hot poultices to the abdomen is of value, since these also tend to relieve the pain. Hot bottles applied to the surface are also useful. Stimulants must often be administered freely and frequently if the stomach is retentive. Ether may be injected hypodermically if necessary. The swallowing of pieces of ice helps to allay thirst and stop vomiting. The loss of water from the system may be supplied by large injections of water into the bowel, or, if necessary, under the skin, as is recommended for Asiatic cholera. Food is not needed for a couple of days, and, if given at all, should be of the most unirritating kind.

CHRONIC ENTERITIS.

Definition.—A chronic inflammation, either catarrhal or ulcerative, of the mucous membrane of more or less of the small or large intestine.

SYNONYMS.—Chronic intestinal catarrh; Chronic diarrhœa.

Etiology.—The disease may be either chronic in nature from the outset or a sequel of the acute form, especially when there have been repeated attacks of this. The causes of chronic enteritis arising in this latter manner are, of course, those of acute enteritis, and need not be enumerated again; and indeed in the enteritis which runs a chronic course from the outset the initial causes are likewise much the same as in the acute form. There are also, however, causative factors to which the chronicity is due. Among these may be mentioned such debilitating diseases as phthisis and malaria, cachectic states in general, and passive congestion of the portal system the result of chronic cardiac, pulmonary and hepatic diseases. It sometimes develops in the course of chronic interstitial nephritis. The presence of any other chronic lesion of the intestine, such as tubercular ulcers or cancer, is attended by a chronic catarrhal process.

The continuance of improper hygiene is a powerful cause. Consequently chronic diarrhœa is common in prisons and among soldiers in camps. It is frequent also in children under two years of age and in the old.

Pathology.—The early pathological alterations are similar to those seen in acute enteritis. Later more characteristic changes take place. The intestine is uniformly or irregularly dilated or contracted. Sometimes the lumen of the tube is greatly narrowed by hypertrophy of the walls. Stenosis is even occasionally brought about by the hypertrophy. The hypersecretion of the mucous membrane and the frequently extensive ulceration produces a serous, mucous, or purulent secretion which covers the lining of the intestine in larger or smaller amounts.

The lower ileum and the colon are the portions usually most affected. The mucous membrane is generally reddish-brown or livid, or finally grayish, as a result of deposition of altered blood-coloring matter. The larger veins are injected. The coloration of the mucous membrane is very irregularly distributed in patches and streaks, some of these being dark red, some gray, some almost black, and some pale and nearly white. The pigment is often deposited most abundantly in the tips of the villi and in rings around the solitary glands or in the centres of these glands. In this way, by the close apposition of very numerous minute black points, the well-known "shaven-beard" appearance of the mucous membrane is produced.

The mucous membrane is thickened and the lymphatic follicles very much enlarged, even more than in acute enteritis. This change is especially well marked in the colon. The thickening is due to abundant small-celled infiltration. There is a hyperplasia of the cellular element of the lymphatic follicles in addition to the serous infiltration which first caused the follicles to enlarge.

Sometimes the mucous membrane hypertrophies so rapidly that polypi are produced. Cysts also form by the blocking up of the intestinal glands. The submucous and muscular layers are also often much thickened by overgrowth of the connective tissue, and the latter by increase of muscular fibre as well; and even the serous coat may exhibit patches of thickening.

Sometimes, instead of thickening, great thinning of the wall takes place, and the mucous membranes and glands are found to be atrophied.

As the case advances countless ulcers begin to form in the lymphatic follicles, especially in those of the descending colon and sigmoid flexure, and even of the rectum. The ulceration is the result of the death and consequent separation of portions of the glands, leaving sharply-defined conical ulcers. These spread in depth and in diameter until the tissue surrounding the solitary glands is also involved. The close approximation of the ulcers gives a sieve-like or honeycombed appearance to the mucous membrane.

The individual primary ulcers are one-tenth to one-fourth of an inch in diameter, but by the confluence of several of them much larger, irregularly shaped ulcers are produced, which may penetrate in depth to the muscular layer of the intestine. Indeed, in rare instances perforation of the intestine may take place. This is sometimes prevented by the formation of adhesions. Healing of the ulceration leaves pigmented areas. Sometimes cicatrization is so extensive that the lumen of the intestine is seriously encroached upon.

Beside these ulcers arising in the follicles, others may develop independently by loss of substance in the epithelial portion of the mucous layer.

Sometimes polypoid growths composed of mucous membrane project from the midst of ulcers. Lardaceous degeneration of the intestinal mucous membrane may develop.

Chronic duodenitis is liable to be followed by thickening of the coats of the duodenum and obstruction to the entrance of bile and of the secretion of the pancreas. Chronic proctitis may be present, with or without involvement of other portions of the bowel. There is found in it great hyperæmia, discoloration and swelling of the mucous membrane, and the entire wall of the intestine may be so thickened that the lumen is much lessened. Ulcers are present, as already described. Periproctitis may attend, even when no perforation has taken place.

Symptomatology.—In the typical form of the disease diarrhœa is the most characteristic symptom. The number of passages varies from one to about eight in twenty-four hours. The diarrhœa is seldom present every day, but is liable to alternate with constipation. Sometimes the evacuations occur only in the morning, while in other cases ingestions of food at any time may cause a passage. The amount passed is variable. It is usually about two to four ounces, but will exceed this in severe cases. The consistence of the stools ranges from watery to semi-solid, and the color may be brown, black, yellow, slate-color, green, or white. The substance passed is fæcal, but often contains portions of undigested food. Very commonly there is present a large amount of mucus, and pus also may be seen; and sometimes one of these constitutes the entire stool. Blood is occasionally visible in the form of bright red streaks, or may produce a black color of the passages when its origin is not in the rectum. The odor of the passages is at times most offensive. Fatty stools sometimes appear, especially in children.

Pain in the abdomen and borborygmi are frequent symptoms. These develop at a variable time after eating or perhaps only just before a passage. In other cases there is merely a sensation of abdominal soreness. Flatulence, with its various forms of secondary distress, is often present. If the stomach be also involved, there are seen such symptoms as loss of appetite, pyrosis, bad taste in the mouth, and coated tongue. Sometimes the tongue is dry, red, and glazed.

There are malaise, apathy, and frequently hypochondriasis or melancholia. In rare cases pronounced insanity may develop, though this may be only the result of a latent tendency called into activity by the lowered vitality and the prolonged reflex irritations. The skin grows pale and emaciation is sometimes extreme. Decided fever is usually absent, except in the later stages, but careful observation will often disclose an abnormally great diurnal variation in temperature. The pulse is rapid, or occasionally slow, and palpitation and dyspnœa often develop on slight exertion. The urine exhibits no characteristic changes, although albumin and casts may be found in bad cases.

In the mild form of chronic enteritis there may be perhaps no diarrhœa whatever, or it may occur but rarely. The combination of constipation with abdominal distention and pain is then the most prominent appearance. The lining of the intestine is coated with mucus, which interferes with digestion and absorption, and which renders the propulsion of the mucus-covered fæces particularly difficult to the atonic bowel. This form is not unusual in children after the period of the first dentition.

The description of the typical form applies to cases in which the entire intestine, or the large bowel with a portion of the ileum, is involved; but, as in acute enteritis, sometimes only certain sections are inflamed. Chronic duodenitis is generally associated with chronic gastritis. It is indicated by the union of icterus, clay-colored passages, and biliary urine with gastric symptoms. The presence of fatty diarrhœa is an additional evidence of duodenitis.

The existence of chronic jejunitis or ileitis is rendered probable by the frequent development of pain in the region of the umbilicus, with flatulent distention, difficulty in digesting starch and sugar, and loss of flesh. Diarrhœa may be absent. If present, the discovery of bile-stained mucus and of undigested food is an indication that the small intestine is involved. There is no way, however, to determine positively the existence of an isolated jejunitis or ileitis. Without doubt, many of the cases generally viewed as "intestinal indigestion," due to functional disturbances and the like, are in reality cases depending on mild chronic inflammation of the small intestine.

Chronic colitis is the form to which the description of symptoms as given especially applies. That the colon is involved is indicated by the large size of the passages, and the greater severity of the pain, which is most apt to develop just before the evacuation of the bowels.

Chronic proctitis is characterized by the frequent desire to evacuate the bowels, accompanied by tenesmus. The stools are often composed largely of mucus or pus.

The symptoms of chronic enteritis vary somewhat with the stage of the malady. When there is no fever or decided impairment of nutrition, and no blood or pus in the stools, it is probable that ulceration has not yet commenced. If, however, pus in the smallest amount or portions of the intestinal mucous membrane, can be discovered, the diagnosis of ulceration is assured. Nevertheless ulceration may exist and give no positive evidence at any time.

Complications and Sequels.—Pneumonia, developing in advanced cases, has repeatedly been observed as a complication of chronic enteritis. Bronchitis and pleurisy may also occur. Peritonitis may result from perforation or may develop without this, and periproctitis may be produced in the same way. General marantic œdema may complicate advanced cases. The various diseased conditions enumerated as causes of chronic enteritis could also be viewed as complications. Ulceration of the cornea has been observed a number of times in cases of camp diarrhœa. Prolapse of the rectum, eczema of the anus, and hæmorrhoids are frequent complications of chronic proctitis.

Among the most important sequels are stricture of some portion of the bowel from cicatricial contraction, although this is rare, chronic constipation from atony of the muscular wall, and persistent intestinal indigestion and consequent malnutrition.

Diagnosis.—The recognition of the disease is generally easy, except in the milder cases where no diarrhœa occurs. The determination of the portion of the enteric tract affected has already been discussed.

Prognosis and Course.—The disease is always very resistant to treatment, of very prolonged course, and especially fatal in children and old persons and those already debilitated by other diseases. The longer it has lasted before treatment is commenced, the more difficult is it to eradicate it. The disease may persist for months or years. It is always intermittent in cases which have lasted a long time, and there are periods during which the patient appears to be recovering.

In cases which terminate fatally emaciation grows extreme, fever is apt to develop, the stools become very frequent and copious, and general cachectic œdema appears. Local œdema may occur, as the result of marantic thrombi. Death results from exhaustion or sometimes from intercurrent pneumonia, bronchitis, thrombosis of the cerebral sinuses, peritonitis, or some other complication.

In cases which recover there is apt to remain a great tendency to attacks of diarrhœa after slight indiscretions in diet or on taking cold.

Treatment.—It is of course necessary to treat any one of the numerous diseased states to which chronic enteritis may owe its origin and persistence. Further, it is important to try to prevent, as far as possible, the development of enteritis in those predisposed to it, by regulating the diet and hygiene.

In the mild cases, in which constipation with evidences of intestinal catarrh prevail, it is necessary to regulate the diet carefully, directing explicitly the kind and quantity of food to be taken in each individual case, and insisting that the food be eaten slowly and chewed thoroughly. As a rule, an excess of starch should be avoided. Sometimes an absolute milk diet followed for a time is an excellent remedy. With this should be combined systematic exercise and massage, bathing, change of air and scene, the frequenting of mineral springs and the drinking of their waters, and other hygienic measures.

Intestinal digestion may be aided by such remedies as pancreatic extract and bicarbonate of sodium. For the relief of the constipation it is frequently necessary to give such mild laxatives as cascara sagrada or small doses of senna, aloin, or podophyllin. Ipecacuanha is often a valuable adjuvant. Small enemata of cold water or the use of small doses of Hunyadi, Friedrichshall, Rubinat, Bedford Springs, or other laxative waters may answer the same purpose. Free purgation is, however, to be avoided. Massage or faradization of the abdomen may be of service for the relief of constipation.

In the severer forms of chronic enteritis, in which diarrhœa is present, dietetic and hygienic regimen, persistently observed, is, if possible, even more important than in the milder cases. Rest in bed is often essential. An exclusive milk diet should certainly be tried, and scraped raw beef is of value in some cases. Starch, fat, and sugar should be avoided. Among the drugs which may be employed, bismuth in large doses holds a prominent place; chloride of iron, tannic acid, nitrate of silver, acetate of lead, and other astringents, as recommended for acute enteritis, are often useful. These remedies act slowly at best, and should not be too hastily abandoned.

Inasmuch as the colon is so especially involved in the disease, local treatment with large enemata is generally advisable. Simple water may be employed, but oftener the addition of some astringent is necessary. Sulphate of zinc (1 to 3 grains to 6 ounces), nitrate of silver (1 to 3 grains to 6 ounces), and acetate of lead (1 to 3 grains to 6 ounces) are among the best. This local treatment is particularly desirable—and, indeed, almost indispensable—when ulceration has clearly commenced. The larger enemata will rarely be retained;

and it is at times better to use small ones, f℥ss or f℥j, containing a small amount of an astringent, which are to be retained.

The employment of alteratives is sometimes demanded, and good results may be hoped for from cod-liver oil and arsenic. Opium should not be given as a routine treatment. It is called for at times in cases in which there is a tendency to frequent attacks of diarrhœa, but it finds little place in the therapy of the severer cases.

The treatment of chronic proctitis is the local therapy already recommended for colitis; that for chronic inflammation of the duodenum is the same as for acute cases.

ENTERITIS IN CHILDREN.

The proneness of young children to diarrhœal diseases, and the variations exhibited in the symptoms and causes as seen in them, render a brief separate account of them advisable.

The diarrhœal diseases of childhood may be divided into—1, Acute dyspeptic diarrhœa; 2, Cholera infantum; 3, Acute entero-colitis; 4, Chronic diarrhœa.

ACUTE DYSPEPTIC DIARRHŒA.

Etiology.—This disease is wonderfully frequent in nursing children, in whom there exists a special predisposition to it, depending upon the organization of the digestive tract. The work of the intestine in children is rendered especially heavy by the small amount of the salivary secretion, the weakness of the digestive power of the stomach, and the deficiency in the strength of the pancreatic juice. Various events in the child's life are liable to be accompanied by diarrhœa and vomiting. Prominent among these is dentition, which, although theoretically normal, is very frequently not so, at least in its effects. Intelligent mothers frequently observe that with the eruption of each tooth an attack of diarrhœa and vomiting takes place, which cannot be accounted for in any other way.

In addition to these causes are general feebleness of the constitution, rickets, and the existence of hot weather. The latter is a very powerful factor; so much so that the vast majority of cases are seen in the summer season. Exposure to cold and wet also aids in producing it, as does improper hygiene of any sort. Particularly to be noticed is the overcrowding, filth, and lack of fresh air and sunshine so common among the poorer classes.

By far the most frequent cause of the disease is a dietetic one. The child is nursed too much; the breast-milk is altered by anger or fright or other emotions experienced by the mother; or the child is given a "taste" of table food or is fed on starchy substances before it has acquired the ability to digest them. Artificially-fed children suffer most, since the diet is more liable to be improper in their case.

The great cause of diarrhœa in bottle-fed children is the abnormal and excessive development of bacteria in the food before or after it is ingested. Normally, there are but few species of bacteria in the stools of an infant, but studies by Booker and others show that in diarrhœa the number of species is greatly increased. It is to the chemical products of some of them and to the alteration of the food that the irritation of the bowel is probably due.

Heat, which has already been referred to as a predisposing cause, acts in most cases by especially favoring the growth of bacteria. In other instances, however, it would appear that the heat itself produces a diarrhœa of relaxation, apart from any action of bacteria.

Pathology.—The lesions of this form of diarrhœa do not pass beyond the stage of catarrh. There is redness, swelling, and increased secretion, but destructive lesions of the intestine do not take place.

Symptomatology.—The symptoms are perhaps ushered in by restlessness at night and a little colicky pain, and then diarrhœa begins. This may be preceded or accompanied by vomiting or may be entirely without it. The passages are increased at first to four, eight, or sixteen in twenty-four hours, and are especially likely to occur after taking food. They are more liquid and more copious than normal, and may be very offensive. Frequently there are few or no passages during the night. In infants the loose stools always contain curdled milk, and often are "yeasty" and of a sour odor. Later the stools become more numerous and greenish, and often of a deep-green color. Mucus is frequently seen, and sometimes a little blood also. Colicky pain with crying often precedes the movements in young children. The urine is scanty, appetite is lost, the tongue is coated, and thirst is increased. There is no fever or but little. Decided wasting may take place in the course of a few days.

Sometimes the attack begins much more abruptly and is more severe. Fever increases rapidly to 104° or 105° F., and nervous symptoms, and even convulsions, are present. These symptoms soon disappear, the temperature falls, and vomiting and purging begin. The passages may become very numerous and watery, but do not assume the characteristics of those of cholera infantum.

All grades of severity of the disease exist between the mildest and the severest cases.

In older children dyspeptic diarrhœa may be brought on by eating green fruit or other injurious substances.

Diagnosis.—The diagnosis is usually easy. The disease is to be distinguished from tubercular diarrhœa, from which it differs in the abrupt commencement and in the absence of any signs of tuberculosis elsewhere in the body. From diarrhœa and vomiting ushering in acute febrile disease it can be distinguished only by watching the course of the case. The difference between acute dyspeptic diarrhœa and entero-colitis and cholera infantum can better be referred to later.

Prognosis and Course.—Attacks of dyspeptic diarrhœa in children last from four to seven days if well managed. Relapses, however, are very prone to occur, especially in hot weather, or a series of attacks may take place at intervals of a week or more, the bowels never becoming perfectly natural between them. Most cases get well, yet enough die in summer to make the disease a serious one. It is especially dangerous in weakly babies, but even in others there is a danger of the disease running into entero-colitis or cholera infantum.

Treatment.—Little more needs to be said of the treatment than has already been mentioned in the discussion of the disease in adults. Most important of all is the attention to the hygiene and the food. It is absolutely necessary in many cases to remove the child from the influence of continued hot weather. Children should be taken to the seashore or mountains when this is possible. When not, as in the case of the poor in cities, they should, at least, be taken during the day into the open squares or parks or on the water. Fresh air is essential, and the patient should be out of doors nearly all day, merely avoiding exposure to the hot sun's rays.

The child should be bathed daily, and in very hot weather several times a day. The clothing should be light and cool. On the other hand, the clothing in winter should be sufficiently warm, and care should be taken that the arms and legs are always well covered. Rest is important in diarrhœal diseases. A sick child should not, however, be kept in the lap in summer, as this but increases the heat.

Diet is of great importance. Both the use of improper food and the introduction of germs are to be avoided. Where it is possible to procure a wet-nurse for an artificially-fed child it is most desirable to do so. In other cases care should be taken that the food is sterilized. Great caution must be observed that the child's stomach be not overloaded. It is indeed better in acute cases to stop all nourishment for twenty-four or forty-eight hours, and to give barley-water or some similar unirritating substance. In many instances it is well for a time to do away entirely with the casein and to feed the child on cream and water, veal broth, albumen-water, or, in the case of older children, on scraped meat or the like.

The medicinal treatment of acute dyspeptic diarrhœa does not differ from that recommended for acute catarrhal enteritis in adults, although especial stress must here be laid on the value of irrigation of the intestine. Intestinal antisepis holds, too, a particularly prominent place in the treatment of diarrhœa in children, since in them especially is the disease apt to be of bacterial origin. Benefit is often derived from such a combination as the following:

℞. Salol,	gr. xv;
Pulv. cretæ. præp.,	gr. xl;
Bismuth subnitratis,	ʒi vel ʒiss;
Pulv. acaciæ,	q. s;
Syr. zingiberis,	f℥ss;
Aquæ puræ,	q. s. ad f℥iv;

Ft. mist.

Sig. Dose (for a child of one year), a teaspoonful every three hours.

Resorcin, naphthalin, hydronaphthol, and benzoate of sodium are also worthy of consideration. Lactic acid has been highly recommended by Hayem. The advisability of the employment of opium must be determined in each individual case. While its use is not to be recommended as routine practice, there are many cases in which it is indispensable, and it may then be used in the form of deodorized laudanum in connection with the above remedies.

CHOLERA INFANTUM.

Etiology.—This disease is an intensely severe form of acute zymotic gastro-intestinal catarrh. It is analogous to cholera nostras of adults in its symptoms and the rapidity of its course. The term should be accurately applied, for large numbers of deaths are attributed to the disease which were really not at all due to it. It is, in fact, a rare affection as compared either with entero-colitis or acute dyspeptic diarrhœa.

The great predisposing causes are continued hot weather, life in cities, imperfect hygiene, the age of the first dentition, particularly that of from six to twelve months and the existence of dyspeptic diarrhœa. The exciting cause is nearly always some trouble with the food.

Pathology.—From a pathological standpoint the disease is really an intense form of acute dyspeptic diarrhœa; for few or no lesions of the intestine are found post-mortem, and never any of an ulcerative nature. The gravity of the affection seems to be due rather to the associated paralysis of the vasomotor nerves of the intestine, with a resulting profuse discharge of serum, than to any violent enteritis even of a catarrhal form.

Symptomatology.—The symptoms may come on suddenly, but oftener are preceded by diarrhœa. The child begins to have copious, acid and often very offensive stools, and these soon become entirely serous and alkaline and leave no stain upon the diaper. The passages may occur every few minutes, and be expelled with force. They contain immense numbers of bacteria with epithelial and round cells. About the same time there develops incessant violent vomiting, which is made worse by any attempt to take nourishment. The vomited matter is at first of food, but later is mucous and bilious. There is often, at the outset, evidence of great nervous excitement, the child moaning and tossing, but soon this gives place to apathy and listlessness. Prostration is extreme from the first; the tongue is dry and pasty, thirst intense, appetite lost. In a few hours the fontanelles become depressed, the eye sunken, the face pale, and the features pinched. The loss of weight may be very rapid. The temperature is generally 105° F. or more, and may continue so until improvement occurs or until death. The pulse grows small, frequent, and finally thready and irregular. The respiration is also irregular and the urine scanty and suppressed.

Diagnosis.—The disease can scarcely be confounded with any other. The diagnosis rests upon the uncontrollable vomiting and diarrhœa, the serous, colorless stools, the extreme prostration and collapse, the high temperature, and the rapid course. These are quite sufficient to distinguish it from acute dyspeptic diarrhœa or from entero-colitis, in neither of which are the stools ever serous.

Prognosis and Course.—The symptoms described may persist for a few hours or a few days and the child recover, or it may die in collapse with coma, convulsions, or high internal temperature. Sometimes the vomiting and diarrhœa cease a few hours before death.

Other cases pass into the algid state of cholera, with subnormal temperature, cold breath, and dyspnœa. In still other cases the passages may diminish and the vomiting cease, but the nervous state denominated "hydrencephaloid" or "pseudo-hydrocephalus" develops. This is characterized by apathy, Cheyne-Stokes or irregular respiration, irregular pulse, pupils contracted or later dilated, retraction of the head, retraction of the abdomen, clutching of the fingers, sunken fontanelles, and a normal or subnormal temperature.

The prognosis of cholera infantum is always grave. It varies directly with the youth of the child, the presence of hot weather, the previous existence of diarrhœa or of other impairment of the health, the use of artificial food, and the severity of the attack. The gravest features are hyperpyrexia, uncontrollable vomiting, intense prostration, and severe nervous symptoms.

In cases which recover the severe symptoms generally begin to ameliorate within twenty-four hours, and the diarrhœa alone remains. This lasts for some time, for recovery is always tedious. Sometimes choleraic symptoms disappear for a few days and then return in full force, and the child dies. In other cases the child may recover from cholera infantum only to pass into entero-colitis and die. Other fatal cases die with the features of the disease in a few hours or a few days.

Treatment.—The treatment of cholera infantum must be most prompt and energetic. The most important indication is the relief of hyperpyrexia, if this exist. The temperature must be taken in the rectum, since the axillary temperature even when determined with care is misleading. A tepid bath should be administered at once, and, beginning with the water at 80° F., it may be cautiously reduced by the addition of cooler water if it seems necessary. It is important to supply the system with fluid to replace that which has been lost. This may be done by the administration of ice or water. Irrigation of the stomach and intestine is very valuable, both to accomplish this end and for cleansing purposes. Stimulants must be used freely, and opium is indispensable. The latter is best given in the form of morphine, and hypodermically, $\frac{1}{100}$ of a grain with $\frac{1}{800}$ of a grain of atropine being a sufficient amount for a child of one year. The dose may be repeated in an hour if needed, and after this as necessity demands. Enemata of from 2 to 4 drops of laudanum in 2 drachms of starch-water are sometimes useful. Bismuth or small doses of silver nitrate in solution are useful internally. If collapse threatens, hot applications or a hot bath may be used; and hypodermic injections of ether or $\frac{1}{200}$ of a grain of strychnine should be administered. The same dietetic and hygienic care is required as has been described as requisite in acute dyspeptic diarrhœa.

ACUTE ENTERO-COLITIS.

Etiology.—This form of the diarrhœal diseases of childhood is a very common and fatal one. It is the scourge of the "second summer." It is commonest in those six to eighteen months of age, and rare after three years. It occurs usually in hot weather and in cities and among the poor, but is not confined to these conditions. The causes are those already mentioned as producing acute dyspeptic diarrhœa. It is also very liable to follow this affection or cholera infantum, and too often is a sequel to the specific diseases of childhood, especially diphtheria, measles, and scarlet fever.

Pathology.—The pathological lesions are situated principally in the colon and ileum, although there may be hyperæmia of the rest of the intestinal tract. The disease might with good reason be called "ileo-colitis." It has sometimes been termed "follicular enteritis," since ulceration of the follicles is present in most cases.

During the first week of the disease the lesions are those of acute enteritis of moderate severity in adults. The mucous membrane of the affected region is swollen and softened, and the epithelium appears to have been shed in some places. The solitary and agminated glands are enlarged and prominent. After a week the solitary glands, particularly in the colon, exhibit ulceration, as do Peyer's patches to some extent. These ulcers rapidly enlarge and deepen, forming a central slough, and penetrate to the muscular layer of the bowel.

In some cases, instead of this change, there is a diphtheritic or pseudomembranous inflammation. This renders the intestinal wall firm, stiff, destitute of folds, and of three or four times its normal thickness. The glandular structure is indistinct and there is no deep sloughing. The wall of the intestine has been rapidly infiltrated with small cells and fibrin in this variety of the disease.

Symptomatology.—As a rule, the disease begins as a dyspeptic diarrhœa, the symptoms of which merge imperceptibly or suddenly into those of entero-colitis. The general condition of a child suffering from moderate diarrhœa appears to become worse. The temperature rises and is continuously elevated, although not often exhibiting the hyperpyrexia of cholera infantum. The stools, too, alter in character. They are small in size, number fifteen to thirty in twenty-four hours, contain considerable mucus, and are streaked with blood. They are usually passed without much pain. Sometimes they are larger in size, and are then less frequent. The bowels are generally moved oftenest in the daytime. The passages are acid, often offensive, and vary in color very greatly. The very small stools are sometimes nearly entirely mucous, and are usually without odor. The characteristic feature of the stools of entero-colitis is the relatively large amount of mucus in them.

Vomiting is not a marked symptom of entero-colitis. The abdomen is distended and hard, and is tender along the line of the colon. Sometimes it is soft and not distended. Appetite is impaired or lost. The urine is diminished in amount and is loaded with urates. Nutrition is impaired and weight very rapidly lost, and, although this does not occur so speedily as in cholera infantum, in cases at all prolonged emaciation it may grow extreme.

The skin becomes loose, dry, and scaly, the features sunken, and the child presents a pitiable appearance.

Diagnosis.—The disease is to be distinguished from acute dyspeptic diarrhœa by the febrile course and the large amount of mucus in the stools, and from cholera infantum by the absence of serous passages, hyperpyrexia and incessant vomiting.

Prognosis and Course.—In one form of the affection the attack lasts from two to four weeks, and then gradually ceases or the child dies. In a second variety the disease passes into a subacute stage. In this form the fever nearly disappears and appetite returns, but diarrhœa persists and nutrition does not improve. The passages number only four or five a day and are large in amount, often offensive, and contain mucus and undigested food. This condition lasts five or six weeks, often with intermissions, and the patient then begins to convalesce slowly. If recovery does not ensue, the disease passes into the chronic form.

In a third—the dysenteric—variety of entero-colitis, which is rather a rare form, the attack is very severe from the beginning. The onset is sometimes sudden, with high fever, frequent passages from the bowels, prostration, delirium, and sometimes convulsions. In other cases of this form the onset is more gradual and the temperature is not much elevated. In both of these forms the passages become very frequent, small, almost entirely composed of mucus, and are passed with severe straining efforts. There is, indeed, nearly persistent tenesmus and colicky abdominal pain. Prostration increases, and death may occur in four or five days or a week; or the child may improve and the case assume the ordinary type of entero-colitis.

The prognosis of entero-colitis is always serious. Its gravity depends upon the youth of the child, the previous health, the method of feeding employed (whether breast or bottle), and the ability to remove the child from such unfavorable conditions as hot weather, city life, and the like. There is always very great danger of relapse.

Treatment.—In the treatment of acute entero-colitis antiseptic drugs hold an inferior place, while local treatment is of the greatest importance. Enemata must be used both for flushing the colon and for their medicinal action. The colon may first be washed out with plain water, and then medicated with from 2 to 6 ounces of a solution of tannic acid (5 grains to 1 ounce), nitrate of silver ($\frac{1}{2}$–1 grain to 1 ounce) followed by a large enema of salt solution or by a mixture of bismuth in mucilage ($\frac{1}{2}$ drachm to 1 ounce). Opium may be added to any of these.

The diet must be most carefully regulated, since there is a special tendency in this form of diarrhœa to the passage of undigested food through the intestine. Veal broth, albumen-water, and cream and water are often preferable to milk foods containing casein.

Chronic Diarrhœa.

Etiology.—Chronic diarrhœa in children (not including that due to tuberculosis of the intestines) does not differ materially from the chronic enteritis of adults. It is a common and very fatal form in infants, and dangerous, though less so, in older children. There are two classes of cases, the first consisting of those in which the intestinal condition is one of catarrh, and the second of those in which it is secondary to acute entero-colitis, and exhibits the severer anatomical lesions which are consecutive to those of the acute disease. We may call all cases of diarrhœa chronic which have lasted over six weeks.

The initial causes of the disease are the same as those of acute diarrhœa. The important factors, however, are those which make an acute diarrhœa persist or bring on an attack which is chronic from the onset. Among these is to be included any defect of constitution, such as syphilis, tuberculosis (not intestinal), rickets, or debilitating influences of any sort. The persistence in imperfect hygiene, hot weather, the use of bad food, and other primary causes tends to make the diarrhœa chronic.

Age is an important factor, inasmuch as children up to the age of two years are much more disposed to it than are those who are older, although sometimes the latter have it and die. The diarrhœa is unhappily often kept up by constantly giving the child to eat whatever indigestible article he chooses to ask for. The persistent irritation thus produced ends in chronic diarrhœa. So also the irritation from the presence of worms may produce the disease.

A series of attacks of acute intestinal catarrh render the patient liable to acquire chronic diarrhœa, and, as already stated, acute entero-colitis may be followed by a severe form of it with grave pathological lesions.

Pathology.—Even in the cases which presented symptoms of only moderate severity during life, and which had been chronic from the outset, there is found, post-mortem, distinct hyperplasia of the lymphatic follicles, especially the solitary glands of the colon. These are swollen and projecting, reach or exceed the size of a mustard-seed, and are surrounded by a small red zone. In more severe cases, especially where acute diarrhœa has preceded the chronic form, there is noticed under the microscope some loss of villi and a decided disappearance of many of the tubular glands. There is a great proliferation of lymph-cells in the mucous layer, with some new-formation of connective tissue in cases in which the disease has persisted a sufficiently long time.

In the worst cases, those following acute entero-colitis, ulcers are found scattered over the mucous membrane, as in the acute disease. Some are seen in all stages of repair.

Symptomatology.—The symptoms of chronic diarrhœa vary considerably. In some cases the disease seems to be chronic from the outset. There may have been dyspeptic disturbances for a long time before diarrhœa commenced, and the passages may have persistently shown undigested food, but may have been not at all softer than normal. Sometimes the stools have been semi-solid, pale, and very offensive. There has been also some colicky pain and abdominal distention, and often tenesmus. Then mucus begins to appear very constantly in the passages, which still, however, may not be more numerous than three or four in twenty-four hours in infants, and may have given no alarm to those in charge. The color of the passages varies, being brown, gray, yellow, or green, and the consistence may be thin or mush-like.

This condition persists for several weeks, while the health of the child has meantime suffered. The stools now gradually become more frequent, equalling ten or twelve a day, and are quite watery, and the general health suffers still more. Sometimes the passages are remarkably fatty.

When the disease follows acute dyspeptic diarrhœa there are no symptoms which certainly indicate that it has become chronic. It is merely designated as such if it has lasted six weeks from the onset.

When acute entero-colitis has passed into the chronic form, the symptoms of acute inflammation disappear, and there is no longer fever, appetite may return, and pain is liable to cease. The diarrhœa, however, persists and the general condition does not improve.

In all cases, if the attack be severe, emaciation and debility finally become great, and a baby a year old may weigh no more than when born. There is much listlessness and pallor; the eyes are sunken; the nasal line of the face well marked; the skin is loose, wrinkled, harsh, and dry; the child is restless, moans a great deal, or whines all the time. The tongue is often red and glazed; the appetite is frequently well preserved; vomiting is uncommon; the fontanelles are depressed; the abdomen is sometimes distended with flatus, sometimes flaccid and retracted. There is seldom fever, and sometimes the temperature is subnormal. The pulse is weak and rapid and the urine diminished in amount.

In children over two years of age with chronic diarrhœa there are irritability, restlessness, night-terrors, perverted appetite, wasting, anæmia, and some degree of prostration. The emaciation is usually not so marked nor so rapid as in infants. There is generally pain with straining at stool, and the passages are thin, offensive, of a brown, clay, or green color, and always contain mucus and undigested food. They are not numerous, seldom exceeding three to twelve in twenty-four hours. Ingestion of food nearly always excites intestinal peristalsis and produces an evacuation of the bowels.

Diagnosis.—The diagnosis is easy in so far as determining that diarrhœa is present. The chief question to be settled is as regards the causes of the diarrhœa, and whether the condition of the bowels accounts for the failure of health. It is also important to determine whether or not ulcerative lesions of the intestine are present. This can be done by considering the nature of the disease which preceded the chronic diarrhœa and by the study of the stools. If prolonged high temperature preceded the development of the chronic affection of the bowels, it is probable that ulceration is present.

It is especially important to separate chronic diarrhœa of the class under consideration from tubercular diarrhœa. In the former the wasting and anæmia follow the intestinal trouble, and are usually in proportion to it; the abdomen is oftener natural or retracted, rather than distended; the spleen is rarely enlarged; and there is no constant elevation of temperature.

In tubercular diarrhœa, on the other hand, there is often decrease in the number of passages without gain in other particulars; the disease occurs, as a rule, after the third year of age; the abdomen is often distended; the spleen is frequently enlarged; there is liable to be enlargement of external lymphatic glands; and fever, although irregular, is probably nearly always present.

Prognosis and Course.—The disease is subject to periods of exacerbation and of temporary improvement. It lasts in children under two years of age from two months to a year. As a rule, severe cases terminate fatally in four months unless convalescence has commenced. Death occurs from exhaustion or from some complication, such as broncho-pneumonia or an acute attack of diarrhœa.

In children over two years of age the disease may last almost indefinitely, sometimes for years, with frequent periods of temporary improvement and of relapse.

The prognosis is always grave, for the disease is a serious one, and especially so when occurring in those under two years of age. It is most serious in those who are the subjects of decided constitutional debility or if there is in action some definite cause which cannot be removed.

The gravity of the prognosis is increased by hot weather, imperfect hygiene, and the like, as in the case of acute diarrhœal disorders. The longer the disease has lasted, the worse, too, do the chances for recovery become.

Treatment.—In the treatment of chronic diarrhœa hygiene and diet are far more important than anything else. Drugs, in fact, hold a minor place. Strict attention must be paid to every detail of hygiene. The disorder may be kept up by improper exposure or inadequate clothing, or unsuitable residence, as well as by indiscretion in diet.

It is impossible to lay down any uniform rules regarding diet. The quantity of food must not be too great. The meals must be regular and eaten slowly. In children who have been weaned all fruit, acids, or indigestible articles must be avoided. Liquid peptonoids often constitute a good addition to the dietary; and pepsin, pancreatin, and pancrobilin are each of value, the last mentioned especially if the stools contain fat. It is not rare that on account of the debility and lowered tone small doses of stimulant, especially of brandy or of sound port wine, are useful.

The condition of the skin requires care. Tepid sponge-baths followed by brisk friction, or, when the systemic tone is greatly reduced, daily inunctions with olive or cod-liver oil, will be found valuable.

Antiseptics are of little use unless there be considerable flatulence or acid gastric fermentation, in which case naphthalin or a powder of bismuth, sodium bicarbonate, and a minute amount of creosote may be used.

Opium and astringents are only indicated during exacerbations or if the passages are large and watery. Under these conditions the combination of salol, bismuth, and chalk (recommended on page 758) may be given with or without opium; or nitrate of silver may be used in solution with opium. As a tonic and astringent the following is at times effective when the above has failed:

℞. Acid. sulphuric. aromatic.,	gtt. xl ;
Morphinæ sulph.,	gr. ½ ;
Elix. simplicis,	f℥ss ;
Aquæ puræ,	q. s. ad f℥iv ;

Sig. Dose for a child of two years, a teaspoonful three times daily in a little water.

Astringent enemata are often of service, especially in the cases consecutive to acute entero-colitis.

It is always important to remember the tendency to lowered vitality. Cod-liver oil will not infrequently exert the happiest influence. Tincture of chloride of iron may be of service both for its tonic and its astringent action. Finally, arsenic in very minute doses is occasionally serviceable through its alterative tonic action or when a malarial taint exists.

PHLEGMONOUS ENTERITIS.

This is a very rare affection which cannot be differentiated during life. It is a secondary process, associated sometimes with strangulated hernia or intussusception. It is also seen at times as a change secondary to typhoid, tubercular, dysenteric, or carcinomatous ulceration. Like phlegmonous gastritis, of which it is an analogue, it consists in a diffuse purulent infiltration or the formation of an abscess in the submucous tissue of the intestinal wall. The pus may penetrate the remaining coats of the bowel. If this occurs through the serous coat, peritonitis follows, or in case the lesion is in the rectum a periproctitis is the result.

PSEUDO-MEMBRANOUS ENTERITIS.

Pseudo-membranous enteritis, also sometimes called diphtheritic enteritis or membranous enteritis, is entirely different from mucous colitis, next to be described, which is likewise often, though wrongly, called membranous enteritis. In the disease under consideration a fibrinous pseudo-membrane is produced in or on the mucous membrane of portions of the small or large intestine.

It may attend some infectious disease, as pneumonia, cholera, scarlatina, pyæmia, tuberculosis, or typhoid fever. It may also be the result of some toxic agency as lead, mercury, or arsenic. It is seen, too, in some cases of entero-colitis in children. Finally, it may occur in the later stages of such diseases as nephritis, cancer, hepatic cirrhosis, or in any cachectic condition.

The pathological lesions found vary in different cases. In some instances there is seen a grayish pseudo-membrane, forming small patches in various parts of the colon and in the cæcum. In other cases a thin yellowish exudation occupies chiefly the tops of folds of the mucous membrane in the colon and ileum, the tissue beneath being much congested. If more extensive, it may penetrate nearly the entire thickness of the mucous layer. The widespread fibrinous infiltration sometimes seen in entero-colitis in children, has already been described. A similar extensive infiltration of nearly the whole thickness of the intestinal wall occurs in many cases of diphtheritic dysentery.

In some instances the solitary glands are the portion chiefly involved and their summits exhibit a diphtheritic necrosis, and follicular ulcers are also common.

The symptoms of pseudo-membranous enteritis likewise vary. None are at all characteristic, and often there are no evidences whatever of intestinal disease. In other cases there are pain and diarrhœa, but straining at stool and the passage of bloody mucus are unusual. Treatment can only be symptomatic, except that directed against the primary disease.

MUCOUS COLITIS.

Definition.—A chronic non-febrile disease of the large intestine, characterised by the production periodically of very tenacious mucus, which is passed in peculiar tubular or membranous forms.

SYNONYMS.—Of the numerous synonyms may be mentioned—Tubular diarrhœa; Membranous enteritis; Mucous disease of the colon; Intestinal casts; Mucous diarrhœa. The term membranous enteritis or pseudo-membranous enteritis is much better applied to the fibrinous condition, often called diphtheritic or croupous, which has just been described.

Etiology.—The disease is not very frequent, and the exciting actual cause is unknown. The affection occurs oftenest in young adult women of a nervous or hysterical disposition. It is liable to attend or follow some other affection of the enteric tract or some disease of the genital organs or nervous system. In a considerable number of cases I have found associated with the disease the existence of small mural fibroids.

Pathology.—It is probable that the only lesion is a low grade of follicular catarrh of localized areas of the colon. No distinct evidences of inflammation can indeed be discovered in some cases. It is clear that the nervous system is implicated in the disease in an important degree. It has even been suggested that the disorder is primarily an affection of the nervous system, with a secondary involvement of the colon.

The mucus passed may be of all grades of tenacity, from a slimy semiliquid to firm, opaque, white membranes. These latter may form a complicated network of cords, or may present a tubular cast of the bowel a foot or more in length, or may be only in small shreds. Both chemically and microscopically these casts are found to consist of mucin and not of fibrin.

Symptomatology.—The disease is a very chronic one. It usually occurs in independent attacks separated by intervals, the duration of which varies much in different cases. During the intervals the patient usually presents evidences of neurasthenia, with more or less pronounced digestive disturbance. Occasionally the course of the disease is continuous, without definite intervals, the membranous discharges being liable to occur at any time. Nervous excitement, anxiety, and fatigue may bring on an attack.

The attack is generally marked by pain in the abdomen, tenesmus, abdominal tenderness, hysterical or other nervous disturbances, and digestive affections. In some cases the nervous phenomena are intense: I have seen hystero-epilepsy and hysterical stigmata precede or accompany every attack in certain cases. Not every evacuation from the bowels during this time will contain the intestinal casts, but some of them do. The symptoms vary greatly in intensity, and in some instances are inconsiderable, while in others the pain is intense. Pain is usually relieved by the passage of the mucus. Blood is sometimes seen in the stools.

Diagnosis.—This is usually made without much difficulty. The membranes are to be distinguished from the other substances, especially intestinal parasites and tendinous parts of the food ingested. Necrotic portions of the mucous membrane of the intestine may somewhat resemble the casts. The history of the case will aid in distinguishing between the two, in addition to the differences which close examination will show.

Prognosis and Course.—The period of aggravation may last but twenty-four hours or even as long as two weeks. The prognosis is entirely favorable as regards life, but unfavorable as regards recovery, the disease being very chronic and not very amenable to treatment.

Treatment.—The treatment must be directed to the restoration of the general health, to the avoidance of attacks, and to the cure of the chronic affection of the mucous membrane of the colon. Regulation of the habits of life; correction of menstrual disorders and relief of uterine or ovarian irritation; improvement of nutrition by rest, travel, or change of residence, tonics and nutrients, especially cod-liver oil—are to be regarded as the basis of the treatment. The avoidance of the attacks demands assiduous care to all hygienic details. The diet should be regulated in such a way as to avoid irritation of the stomach and bowels, and constipation should be prevented. At the same time it is often found best not to secure a movement oftener than alternate days or even twice a week. Large enemata of water, about one quart, containing nitric acid, gradually increased from 15 drops to 25 or even to 50 drops, have seemed peculiarly useful. Counter-irritation over the colon by repeated light applications of the thermo-cautery is to be advised.

Internally, hydronaphthol in the form of keratin-coated pills; naphthalin; oil of turpentine in emulsion; unirritating preparations of iron—full doses—have proved valuable in various cases.

During the attack the treatment must be symptomatic. Asafœtida, monobromide of camphor, cannabis indica, phenacetin, may be found useful. Opium often seems indicated, but the danger of establishing the opium habit makes strongly against its use.

APPENDICITIS.

Definition.—An inflammation of the vermiform appendix.

Etiology.—This affection is a comparatively common one. It is certainly far more frequent than stercoral typhlitis, presently to be described, as it is also far more serious.

It is a disease most liable to occur in young persons, Fitz's statistics showing that about 50 per cent. of the cases are in those under twenty years of age. It is also much more common in males than in females.

Injury resulting from overstraining or from a blow appears to have produced the disease in many cases. Constipation is an important cause, and indiscretions in diet occasion it in other instances. Sometimes typhoid or tubercular ulceration, or rarely catarrhal ulceration, may produce it.

The most frequent cause, however, is the entrance into the appendix and retention there of irritating fæcal matter. Normally the appendix is closed by a valve, but in cases in which the valve is imperfect or the appendix unusually broad or abnormally situated fæcal matter or foreign bodies may enter it.

Although a large variety of foreign bodies have been found in the appendix, yet it is rare that these are the cause of inflammation as compared with fæcal concretions. These concretions consist of inspissated fæces and mucus with lime salts, and are often of almost stony hardness. They are oval in shape, and often have much the form of a date-stone. Sometimes a small foreign body occupies the centre of one of these secretions.

Pathology.—The lesions differ according as the process is or is not an ulcerative one. In simple catarrhal appendicitis the process does not go beyond the catarrhal stage. The mucous membrane is swollen and covered with mucus, the submucous and muscular layers are also thickened and rigid, and the serous layer is inflamed, so that adhesions form with surrounding parts. The appendix may attain the size of an adult finger. In its cavity there are usually one or more concretions. This form of appendicitis may terminate in resolution, but is more liable to go on to the ulcerative form.

In ulcerative appendicitis an ulcer forms and may go on to perforation, but this rarely happens except when the process is due to the presence of a concretion or a foreign body. If the ulcer cicatrizes without perforation, the shape and structure of the appendix may be eventually greatly changed, and the organ may, indeed, be nearly entirely obliterated.

The perforation of an ulcer may be followed by various results, according to the position of the appendix. If it has not become attached by adhesions to surrounding parts, but is freely movable in the peritoneal cavity, perforation will be followed at once by an intense purulent peritonitis. This is not the usual rule, for an adhesive peritonitis has generally preceded the rupture of the ulcer. In such cases a circumscribed perityphlitic abscess is produced. The quantity of pus may be very small and may be surrounded by a large amount of inflammatory tissue. Such a condition may end in the absorption of the pus and in resolution. In other cases a large abscess forms and ruptures in some direction. If rupture takes place into the peritoneal cavity, as is often the case, diffuse violent peritonitis results. Generally such an abscess opens into the intestine, but it may rupture through the abdominal wall, or into the bladder, pleura, portal vein, iliac artery, or elsewhere.

If the appendix be situated posteriorly, the suppurative inflammation following ulceration will soon extend into the retroperitoneal tissues and form a paratyphlitic—*i. e.* a retroperitoneal—abscess. As in perityphlitis, this may extend in various directions, but especially downward toward Poupart's ligament or upward toward the kidney.

Symptomatology.—The symptoms of appendicitis are not always alike. The simple catarrhal cases may produce very dangerous symptoms, and, on the other hand, abscess may result and yet symptoms be sometimes largely latent. So, too, cases which begin with mild symptoms may suddenly end fatally, and in others which commenced with great severity the inflammation may stop at almost any period of its course.

In typical cases—so far as any case can be called typical—there may be prodromal symptoms, consisting of localized colicky pain and tenderness, alternating constipation and diarrhœa, and possibly nausea and vomiting; but, as a rule, the onset is rapid. There suddenly develops intense abdominal pain, which may be at first diffuse, but is often from the beginning localized in the right iliac region, which is made worse by every movement of the body or by the slightest pressure, and which compels the patient to walk bent forward and leaning to the right, or, if still more severe, to lie in bed on the right side with the right leg drawn up. The pain in the iliac fossa is constant, although attended by exacerbations in its intensity.

Fever of irregular type commonly soon develops, and the pulse is accelerated and hard. The respiration may be embarrassed or superficial as a result of the pain. There may be an initial chill. The features express pain and anxiety. Hiccough is sometimes annoying; vomiting is common; thirst incessant; appetite lost; and obstinate constipation the rule. The urine is diminished in quantity and highly concentrated.

Physical examination of the abdomen shows it to be distended or hard and flat. There is generally great tenderness in the right iliac fossa. In some cases this is particularly marked at what is called McBurney's point—viz. one and a half to two inches from the anterior superior spine of the ileum in a line drawn from it to the umbilicus. In twenty-four to forty-eight hours or longer a swelling often appears in the region of the cæcum. Sometimes it is sharply defined, in other cases diffuse, and in a large number the tension and tenderness of the abdominal walls, or, if the tumor be small, the intervention of distended coils of intestine, prevents its being detected. In such cases etherization may be of advantage, or the mass may be within reach of the finger in the rectum. The tumor as felt through the abdominal walls may be nodular or smooth, elastic or hard. Percussion is unaltered if the mass be small, but dull-tympanitic if it be large or near the surface.

Course.—In the mild cases in which resolution takes place or the pus is small in amount and may be absorbed, the pain ameliorates, the fever ceases, and constipation yields after a few days, and the induration and tension disappear entirely in about two or three weeks.

In more severe cases, where such a termination is not to be hoped for, a sudden evacuation of pus through the bowel may be attended by rapid disappearance of the threatening symptoms, or the abscess may at length find a vent through the abdominal walls. In other cases it is not discharged in this way, and various septicæmic symptoms develop.

The greatest danger is that of perforation into the peritoneal cavity with the development of a diffuse purulent peritonitis. This may occur at any time after pus has formed, but does so oftenest during a period extending from the second to the fourth days of the disease, inclusive. Such an accident is indicated by the aggravation of all the symptoms with the development of diffuse abdominal pain, tenderness, tympanites, and other evidences of inflammation of the peritoneum. Such cases very rarely recover.

Diagnosis.—An affection presenting the symptoms of severe pain in the right iliac fossa coming on suddenly in persons previously well, with tenderness on pressure in this region, induration, vomiting, and constipation or diarrhœa, is probably appendicitis in nearly every instance.

Stercoral typhlitis has a less abrupt onset, slight pain, less severe general symptoms, and the presence of a doughy mass in the right iliac fossa, which region is less tender to pressure. The symptoms are promptly relieved if the bowels can be well opened by a laxative. Nevertheless, the diagnosis is often impossible, and it is probable that many cases called typhlitis are in reality instances of mild appendicitis.

Perinephritic abscess cannot be definitely distinguished from paratyphlitic abscess resulting from appendicitis, unless it be by the history of the case and by the discovery of some abnormality in the urine.

Invagination and strangulation may simulate appendicitis closely unless the whole course of the case be known. In intestinal obstruction there are likely to be fæcal vomiting, pain, tenderness, and sometimes a tumor, all situated in some other region than in that of the cæcum; and in intussusception bloody stools with tenesmus occur.

To distinguish the varieties and the stages of appendicitis is often not possible. The catarrhal exhibits in general a milder course than the ulcerative form, but to this statement the exceptions are too numerous to allow to it much value. As a rule, the onset of the sudden pains marks the occurrence of ulceration through the appendix, and the symptoms which follow are those of the localized peritonitis produced. The improbable suggestion has been made that in certain cases this sudden pain marks the time when some foreign body or intestinal concretion is forced from the cæcum into the appendix, and that the pain is analogous to that experienced during the passage of a gall-stone.

Prognosis.—It is exceedingly difficult to determine the prognosis of the disease in general, and equally so to estimate the chance of recovery in any individual case. The affection is certainly a very grave one, although probably the majority of cases will recover, even without surgical treatment. The disease is dangerous in every case, since there are no sure criteria by which one can predict whether the inflammation will be severe or slight, and whether or not surgical interference will be required. Cases which do not pass beyond the stage of catarrhal appendicitis with adhesive peritonitis always get well. If the process is and remains one of localized peritonitis with slight suppuration, but with no evidence of general septic poisoning, the prognosis is good. Cases in which an abscess perforates into the bowel offer a fairly good prognosis, while those in which the opening is through the abdominal walls are still more favorable.

The prognosis is very unfavorable where the pus enters the general peritoneal cavity or where an acute septic inflammation spreads rapidly in any direction. It is favorable where acute symptoms subside in the course of a few days and the tumor begins to disappear. There is risk, however, as long as any induration remains.

In about one-half the number of fatal cases death occurs within a week after severe symptoms develop. Prompt surgical interference in suitable cases renders the prognosis much more favorable.

Appendicitis shows a great tendency to recur in those who have once had an attack; and this, too, adds to the gravity of the affection, since some one of the attacks may be fatal.

Treatment.—Probably no disease requires more judgment for its proper treatment, and few subjects have been more combatively discussed than the question in what this treatment should consist. So far as medical treatment goes, the great standby is opium in full doses, combined with absolute rest and the application of cold or of leeches to the cæcal region. The much-talked-of danger of "masking the symptoms" by the use of opium undoubtedly exists, yet statistics as certainly show that the mortality has been greatly lessened by this method of treatment.

Enemata of warm water may be used to relieve constipation, but it is questionable whether the rectum and colon should not share in the rest desired in well-developed cases of appendicitis.

For the same reason that opium is to be recommended purgatives are to be condemned. The disease, when at all in a condition for medicinal treatment, is a localized peritonitis, perhaps with an abscess already forming. The administration of purgatives disturbs just that rest of the bowel which is so earnestly to be desired, and greatly increases the danger of rupture of the abscess and the production of a general peritonitis. It is to be borne in mind that neither stercoral typhlitis nor general peritonitis is now under consideration, in each of which conditions saline purges are of value.

Yet, although the pus produced by perforating appendicitis may be absorbed in a great number of cases, the uncertainty about the chance of this in any given instance or about the outcome of any grave case makes the disease really a surgical affection in the majority of instances. The duty of the physician is to determine in what cases and at what time operative interference should be made. Although no absolute rule can be laid down for determining this, yet, in general, operative measures should be promptly taken whenever acute and increasingly severe symptoms, such as abdominal distention, rapid high pulse, and fever, with localized tenderness and pain, indicate the presence of pus about the appendix, even although no tumor or induration can be detected. The matter of the greatest importance in this connection is that the operation be performed early, before pus has had a chance to perforate the peritoneum or to burrow in various directions.

In cases where there is a great tendency to the recurrence of severe attacks, the operation should be performed, since not only do these attacks seriously interfere with the patient's comfort and occupation, but there is always danger that some of them may prove fatal. The operation may be done either during one of the outbreaks of the disease or in the interval between them. On many accounts it is best to wait until one of the attacks has come on.

TYPHLITIS.

Definition.—An inflammation of the cæcum.

SYNONYMS.—Cæcitis; Typhlo-enteritis; Typhlitis stercoralis.

Etiology.—This affection, formerly considered very common, is now deemed much less so, since the view has become so generally accepted that the majority of cases of acute disease in the right iliac fossa are in reality appendicitis. The terms perityphlitis and paratyphlitis should not be used as designating distinct diseases, since each condition is but the result of perforating appendicitis in almost every instance.

Little positive is known of typhlitis, inasmuch as the majority of cases recover. Many of the cases called typhlitis may have been, and probably often were, instances of appendicitis.

The disease occurs chiefly in young persons, and oftener in males than in females. Those who eat much or improperly and exercise little, and who have a tendency to constipation, are rather more disposed to it. Over-exercise and exposure to cold may likewise aid in the development of the disease. The active cause, however, is the arrest of fæcal matter in the cæcum, since this, like every foreign body, acts as an irritant and sets up inflammation.

Although typhlitis may occasionally result from the presence of tubercular, dysenteric, typhoid, or cancerous ulcers or from the action of some sharp foreign body, yet so usually is fæcal accumulation the cause that the common term "typhlitis stercoralis" may practically be considered as synonymous with typhlitis.

Pathology.—As the disease ends in recovery in nearly every instance, the pathological lesions are not well known. As described, they are similar to those of catarrhal inflammation in other portions of the intestinal tract, with thickening of the intestinal walls, but usually without ulceration. If the inflammation be very severe, the serous layer may be involved and a localized adhesive peritonitis form. It is possible for a fæcal abscess to develop and for perforation of the cæcum to occur, with escape of pus and fæces; but such a termination is unusual.

The lesions of typhoid, tubercular, dysenteric, and cancerous forms of typhlitis are practically the same as those produced by these diseases in other parts of the intestine.

Symptomatology.—The symptoms are much as in appendicitis, but less severe. Though they sometimes come on suddenly, there are liable to have been digestive disturbances for several days. There develop pain and tenderness in the right iliac fossa; a tendency to flex the right thigh, which is worse if the inflammation spread to the peritoneal coat; constipation, slight fever, and sometimes vomiting. Palpation generally reveals a tumor in the position of the cæcum, which is sausage-shaped and can be indented by the finger if tenderness be not too great to permit of this. The abdomen is commonly much distended by the accumulation of gas and of fæces. Sometimes there is complete intestinal obstruction with fæcal vomiting, but this occurs only in bad cases.

Diagnosis.—The disease must be distinguished from appendicitis, and the points of difference have already been given in the discussion of that affection. In many cases no positive diagnosis can be made.

Prognosis and Course.—The prognosis is good, inasmuch as nearly every case recovers. Flint states that he never saw an acute case end otherwise than in recovery. In some instances, however, death may result with symptoms of intestinal obstruction or from perforation of the cæcum.

Ordinarily the fæcal mass is expelled from the cæcum after a few days or a week, and the symptoms very rapidly disappear. There is, however, a great tendency to the recurrence of the affection.

Treatment.—The patient should be at complete rest in bed, with ice or poultices applied to the iliac region. Enough opium should be given to relieve pain. So soon as the signs of acute localized peritonitis, if such be present, have been subdued, the effort should be made to start again the passage of the intestinal contents through the cæcum. For this purpose large enemata may be administered three or four times a day through a long flexible tube. In many cases good results may be obtained by the administration of mild laxatives, especially castor oil, Rochelle salts, or divided doses of calomel. If irritability of stomach exists, food must be withheld and all laxatives avoided save minute doses of calomel. In the most serious cases, where there is reason to believe that perforation has occurred, operative interference should at once be adopted.

DYSENTERY.

Definition.—An inflammatory disease of the large intestine, often, and perhaps always, of an infectious nature, characterized by catarrhal, ulcerative, and pseudo-membranous lesions, and by the discharge from the intestine of mucus, blood, and pus, with much straining and severe colicky pain.

General Remarks.—Dysentery may, with good reason, be considered a group of symptoms rather than a distinct disease, since not only do the pathological lesions vary greatly in different cases, but the symptoms, and even the causes, seem to an extent diverse.

Even if it be admitted that the disease is in every case an infectious one, there is no knowledge of any one germ which always produces it. On the other hand, it seems likely that the *amœba coli*—the probable cause of many cases of epidemic tropical dysentery—is not the invariable producer of the disease in all the sporadic or other epidemic cases, and that many of the former, as well as the instances of secondary diphtheritic dysentery, are not of an infectious nature at all.

As regards the question of classification, it is impossible to adopt any which is at all satisfactory, although various ones have been proposed by different writers. The difficulty consists in the fact that the pathological and clinical classifications do not correspond. We may most conveniently divide the disease from a clinical standpoint into *sporadic*, *epidemic*, and *chronic dysentery*, while from the standpoint of pathological anatomy we can divide it into *catarrhal*, *amœbic*, *pseudo-membranous* or *diphtheritic*, and *chronic* forms. But sporadic cases, although usually catarrhal, may sometimes exhibit the gravest anatomical lesions, while many epidemic cases are mild both in symptoms and pathological changes.

Many other varieties of dysentery have formerly been described, but as these indicated only special features in certain cases or the existence of complications, they do not require a separate consideration. Among them may be mentioned the bilious, typhoid, intermittent, rheumatic, hæmorrhagic, algid, and gangrenous forms.

Etiology.—A number of factors may be considered as predisposing causes. The subjects of the disease may be of any age, but adults are most frequently attacked. More males than females suffer, since the former are more exposed to the influences which produce the epidemic form.

Climate has a powerful action in producing it. The affection is essentially one of hot climates, for, although it may occur sporadically, and sometimes even epidemically, in northern countries, it is in the tropics that its greatest ravages have been made. The season of the year, too, is of importance, as shown by the fact that much the greater number of cases occur in the summer and autumn. Sudden changes in temperature also appear to possess etiological power, as does the presence of decaying animal or vegetable matter. Imperfect hygiene of any kind is commonly claimed to aid in the development of dysentery. Thus, filth, exposure to cold, overcrowding in camps or prisons, excessive fatigue, and the like are all of decided influence.

The use of poor or insufficient food and the ingestion of unripe fruit and other articles which disturb the digestion seem sometimes to predispose to the disease. So, too, the drinking of stagnant or impure water has long been recognized as an important cause.

It often seems that the nervous system plays a distinct part, and that mental depression may predispose to the development of dysentery. For this reason, as well as on account of filth and starvation, the occupants of war-prisons have been particularly liable to the disease.

Besides being the result of the causes mentioned, dysentery may occur as a secondary affection,—a sequel to, and often a fatal termination of, typhoid fever, typhus fever, chronic nephritis, pyæmia, pneumonia, scarlatina, variola, tuberculosis, cancer, chronic heart disease, scurvy, and cachectic states in general.

It is not certain whether any of the agencies mentioned as causes of acute primary dysentery have more than a predisposing power and can themselves act as exciting causes, or whether they simply favor the development of the specific germ. It is certainly probable that sporadic cases may be produced directly by some of them, and that there is no specific germ for this class of cases. On the other hand, the remarkable disposition of the disease to occur in epidemics is exceedingly suggestive evidence of the infectious nature of this form of the disorder. Armies especially have repeatedly been the victims of widespread epidemics. Epidemic dysentery has probably killed more soldiers than all other diseases put together, and in the civil war of the United States was the cause of death of probably over three hundred thousand men. This simultaneous development of a large number of cases in one locality is almost convincing proof that there must be, at least in certain forms of the disease, some specific germ, which is most naturally to be sought for in the fæcal evacuations.

Although other micro-organisms have been described by some writers, that which is now generally considered as the cause of epidemic tropical dysentery is the amœba coli. Lambl described it in 1859, and Loesch in 1875. The latter considered it to be the cause of the disease, and was able to produce symptoms of the affection in a dog by injecting the dysenteric stools into the intestines of the animal. Since that time the organism has been repeatedly found by Koch, Kartulis, Osler, Councilman and Lafleur, Dock, and others. There seems to be little doubt that the amœba coli is constantly present in the stools in many cases of epidemic tropical dysentery, and it is most probable that it may be looked upon as the specific cause of these cases, although the claim has been made by Massiutin that it may be found in other intestinal diseases as well.

The organism is an unicellular, motile body, measuring 20 to 50 micromillimetres in diameter, and consisting of granular protoplasm which contains a nucleus and several vacuoles. Not only is it present in the evacuations, but it has been found as well in the pus from hepatic abscesses secondary to dysentery.

The manner in which infectious dysentery is propagated is a question of interest. It seems probable that the germs have a decided tenacity of life. It is also probable that they are disseminated by means of drinking-water.

Whether the disease is contagious is an unsettled question. Although there are cases on record which point strongly to its communicability, the mass of evidence shows that it is certainly not often contagious.

Pathology.—The lesions found in the intestine after death from dysentery are very unlike in different cases. Although they do not differ materially from those already described under some of the forms of diarrhœa, it is well to review them here for the sake of completeness of discussion.

The milder cases, from a pathological standpoint, exhibit the lesions which go to make up catarrhal dysentery, while the cases with severer lesions constitute ulcerative and diphtheritic dysentery. Most of the epidemic cases exhibit the severer lesions, while the sporadic cases are generally of the catarrhal form.

In catarrhal dysentery the large intestine is the portion almost solely involved, and particularly the cæcum, the hepatic, splenic, and sigmoid flexures, and the rectum. Sometimes the lower part of the ileum is also attacked. Areas of the mucous membrane are hyperæmic, swollen and covered with tenacious, yellowish or brownish-red mucus, with which some pus is at times mixed. Punctate or diffused extravasations of blood are usually visible. The solitary glands are much enlarged, and project as grayish-white elevations, from the size of a pinhead to that of a pea. The submucous tissue is swollen, and sometimes more so than the mucous membrane. This swelling is due to congestion and serous infiltration.

Sometimes the force of the disease seems to have been expended principally upon the follicles, producing follicular dysentery.

If the process be a severe one or the disease prolonged, small ulcers form about the apices of the follicles (follicular ulceration). The process does not often go beyond this in the sporadic cases, but in the graver forms, especially in epidemics, the follicular ulcers may rapidly deepen and spread, and the surrounding tissue also ulcerates (catarrhal ulceration). This widespread ulceration is found in the cases dying from amœbic dysentery. The ulcers are irregular in shape with undermined walls, and often with a small opening as compared with the size of the cavity beneath. They may penetrate in depth even to the serous coat of the bowel, and may be so numerous that but little unaffected mucous membrane remains. An inflammatory cellular infiltration occupies the tissue immediately around the ulcers, and in this region the destructive process spreads. The walls and floor of the ulcers contain few or many amœbæ. There is a notably small amount of pus present.

In very many cases, particularly of the epidemic variety, pseudo-membranous changes are to be found post-mortem, whence the title "diphtheritic dysentery." When this process is not very severe, patches of thin yellowish or grayish membrane here and there coat the surface of the folds in the colon. In graver cases the whole of the mucous lining of the colon may be involved, and the diphtheritic infiltrate may penetrate the entire thickness of the intestinal wall. The bowel is then enlarged and its walls stiff and thick, and the mucous membrane is transformed into a tough, yellowish substance which consists of an exudation of fibrin, pus, and blood, in which the glandular cells are not visible. The mucous membrane, thus in a state of necrosis, sloughs, and even the submucous tissue and the muscular wall may share in the process. The sloughs, which are sometimes of great size, come away after some time, leaving irregular ulcers of the appearance already described. These ulcers may occasionally perforate.

As previously stated, diphtheritic dysentery may develop as a terminal complication of several other diseases. In this case, as in the primary form, there may be simply a thin coating of pseudo-membrane deposited on the mucosa, or the entire mucous membrane may be necrotic throughout.

If the ulcerative process has been very extensive, death is almost certain to occur. In the event of recovery the ulceration cicatrizes and stricture of the intestine may be produced, but this is very unusual. The healing of the ulcers is very slow, the case meanwhile exhibiting the symptoms of chronic dysentery.

In any case which has lasted long enough to constitute chronic dysentery the mucous membrane becomes more or less pigmented. This pigmentation may be dotted in the centre of the lymphatic follicles and produce the "shaven-beard" appearance. There are also visible scattered patches of steel-gray or blackish color. The mucous membrane is rough and irregular, and may present numerous puckered cicatrices. Every stage of the process of ulceration is also generally present, and the ulcers are much pigmented. Cystic degeneration of the glandular cells of the mucous membrane may be visible. The submucous and the muscular tissues become greatly hypertrophied.

Of other parts of the body affected, the liver is the most important. Single or multiple abscesses frequently occur, especially in cases of tropical dysentery. The contents of such an abscess consist of necrotic tissue with but a small amount of pus. The abscess-walls may be soft and necrotic or, in the older cases, hard and fibrous. Perforation of an abscess into the lung not infrequently occurs.

Besides the abscesses there are scattered foci of necrotic tissue throughout the liver. The mesenteric glands are usually swollen.

Symptomatology.—Although the disease sometimes begins with great suddenness, in the majority of cases it is preceded by an ordinary diarrhœa, perhaps with some malaise, abdominal pain, and evidences of indigestion. There is seldom a pronounced chill. This condition may last twenty-four hours or even several days, and then the characteristic symptoms of dysentery develop. These consist of an altered character of the stools, "tormina" or griping abdominal pain, and "tenesmus," or the constant disposition to strain in the ineffectual effort to empty the bowels.

The dysenteric stools are at first partly fæcal and partly mucous, but very soon consist almost entirely of mucus in the form of jelly-like masses, alone or mingled with blood or pus. Feculent matter is now not passed at all or only occasionally in the form of small scybalæ. Blood, which, like mucus, is very characteristic of the dysenteric stool, is sometimes evacuated in large quantities, even in the absence of any large erosion of a blood-vessel. Ordinarily, however, the blood is moderate in amount and is mixed with mucus.

In cases of diphtheritic dysentery shreds and larger portions of pseudo-membrane or of sloughing tissue are present in the stools. In rare instances even a fibrinous cast of a portion of the intestinal tube may be evacuated. When large sloughs are passed the stools have a dark color and a gangrenous odor.

In severe cases of epidemic dysentery the passages, instead of being chiefly mucus, very often consist of a sero-sanguinolent, highly albuminous discharge, which is composed of blood mingled with exuded blood-serum, and in which float minute pieces of membranes or of sloughs, so that it resembles the scrapings from raw meat. These passages have a very offensive odor.

The dysenteric stools are usually alkaline in reaction. They are of small size, but very frequently number thirty or forty, and even up to two hundred, in twenty-four hours. As the patient improves the number of passages grows less, the amount of blood is diminished, some of the stools are greenish and of a mush-like consistency, fæcal matter begins to appear, and finally the passages are again purely fæcal.

The tormina of dysentery constitute one of the earliest of the symptoms. They consist of peculiar, severe, twisting or griping pains located in the colon. They are paroxysmal in their occurrence, usually worse before an evacuation of the bowels, and worse also toward evening. Pressure on the abdominal walls gives some relief. There is some tenderness on pressure over the course of the colon, but this is usually slight.

Tenesmus is a very characteristic symptom and a cause of great distress. It is due to the inflammation in the rectum, which produces a sensation as though this part of the bowel were constantly full. Frequently the patient feels as though there were a red-hot iron in the rectum. There is a constant, irresistible desire to empty the bowels, and the effort is accompanied by very severe straining, yet the passage of mucus present in the rectum gives no relief, and the patient continues to strain ineffectually until exhausted.

The intensity of the tormina and tenesmus is not always in proportion to the gravity of the case, since even in fatal cases they may sometimes be absent throughout.

In addition to these characteristic symptoms others attend the course of the affection. The temperature is somewhat elevated, but is only exceptionally high. The pulse is not much accelerated except in the gravest cases, where it may be rapid and weak, although it still may sometimes be without increase in velocity. The tongue is coated and moist, or is natural in appearance, or later in the disease may be dry, red and glazed. The abdomen is not usually distended, and may be flat and hard. Nausea and vomiting do not often occur in sporadic cases. In some of the epidemic cases, however, they are most frequent. Appetite is impaired and thirst is very often great. The intellect is usually unaffected or only clouded near the end of life. In some instances, however, delirium is a marked symptom. Strangury sometimes attends the rectal tenesmus, and but a few drops of urine are passed from the bladder, perhaps with the admixture of blood. In severe cases, especially in epidemics, the urine may contain albumin and casts.

As the disease progresses the strength of the patient is greatly diminished, and extreme emaciation may occur as the result of the great loss of albumin in the stools. The typhoid state may sometimes develop. In fatal cases an algid stage, much like that of cholera, often appears, and this condition of collapse may last for several days before death occurs.

The symptoms as detailed are not all found with equal severity in every case. There are, besides, clinical differences between the various forms of the disease mentioned, although these are far from absolute. In most of the cases of sporadic dysentery—*i. e.* in those corresponding to a catarrhal state of the intestine—the symptoms come on rather suddenly after a premonitory diarrhœa, and are milder than in the epidemic cases. The stools usually continue to be composed chiefly of mucus mingled with some blood, the tenesmus and tormina are marked, and nausea and vomiting are unusual.

In the epidemic form and in some of the graver sporadic cases the onset may be very gradual, or there may be, from the beginning, very characteristic symptoms of dysentery. Sometimes the attack commences so suddenly that it seems like a case of poisoning.

In the numerous epidemic cases in which there is an extensive primary pseudo-membranous inflammation in the bowel the patient seems very ill from the onset. The temperature is high, the pain severe, the stools very numerous, prostration great, and delirium and the typhoid state are liable to develop. Sometimes the passages remain diarrhœal in character, and tenesmus may not be marked. In such cases the disease resembles typhoid fever very closely. Many severe cases of epidemic dysentery are characterized by the sero-sanguinolent discharges described, in which shreds of diphtheretic membrane float. Such passages always indicate that the disease is severe.

Tormina and tenesmus are more liable to be wanting in epidemic dysentery than in the sporadic form, and there is, in fact, often less suffering in the former variety of the disease. Nausea and vomiting are, however, more apt to occur.

In the dysentery due to infection by the amœba coli the onset may be gradual or, in severe cases, sudden. The temperature is not much elevated, and sometimes not at all so. Tormina and tenesmus may be entirely absent, although usually present at the outset. The stools are irregular in frequency and character. They may be bloody and mucoid as described, but are more likely to be fluid, from six to twelve in twenty-four hours, and of a yellowish-gray color, although containing some mucus and frequently blood. These cases run an irregular and prolonged course with periods of temporary recovery.

In the diphtheritic dysentery secondary to other diseases there are seldom symptoms characteristic of the pathological change present. There is usually some diarrhœa, with perhaps a small amount of mucus and blood at times.

Chronic dysentery may be chronic from the beginning, but, as a rule, follows an acute attack. Tormina and tenesmus are often absent altogether. The passages vary in number from three or four to twenty or more in twenty-four hours, and are thin and frothy, partly fæcal and partly mucous, and contain undigested food. Blood and shreds of tissue are not often present. Sometimes constipation alternates with diarrhœa. Flatulence is common, digestion disordered, the appetite variable, and the tongue usually like raw beef. There are liable to be exacerbations, during which the character of the stools becomes more dysenteric and blood appears in them. Emaciation and loss of strength are generally very great.

Complications and Sequels.—Dysentery is subject to various complications and sequels, although the majority of the cases escape them. Many of the varieties of dysentery which have formerly been described are simply instances of the presence of complicating diseases. Intermittent dysentery, for example, is an instance of this, the disease being complicated by malaria. The association of these two disorders has been often seen during widespread epidemics. Neither malady appears to exert any influence upon the course of the other.

Typhoid fever may exist in combination with dysentery, and many of the cases supposed to be instances of the typhoid state in dysentery are in reality examples of the coincident occurrence of the two diseases. This combination is especially frequent in certain epidemics; it is rare in sporadic dysentery.

Typhus fever also has been associated with dysentery.

A rheumatic dysentery has been described, but it is not certain whether the arthritis is of rheumatic nature or whether it is allied to gonorrhœal arthritis. In any case, arthritis, especially of the knee, but occurring usually in several joints at the same time, is a very common complication. It appears, as a rule, in the second week of the disease or during convalescence, and has been seen with frequency in some epidemics. Its occurrence and its severity do not seem to bear any relation to the gravity of the attack of dysentery. It lasts four or six weeks.

Dysentery and scorbutus are sometimes associated in those exposed to the causes of each.

Pleurisy, pericarditis, endocarditis, and manifestations of pyæmia are occasional complications. Parotitis and venous thrombosis sometimes occur. Anæmic dropsy may develop in cases which have been prolonged.

Peritonitis is a complication which may result from perforation of the intestine, or more rarely develop from simple extension of inflammation, without the occurrence of perforation. Perforation in the region of the cæcum sometimes takes place, and produces perityphlitis, while that in the lower part of the rectum is followed by periproctitis. Intussusception or prolapse of the rectum may develop in children as a result of the straining efforts.

Abscess of the liver is a serious complication which is very common in tropical countries, but much rarer in temperate climates. It is estimated that it occurs once in every four or five cases of dysentery in India.

Paralysis due to neuritis, and usually represented by paraplegia, is an occasional sequel. Stricture of the colon is a very rare sequel. Persistent irritability of the intestine is a very common sequel of a prolonged attack of dysentery; and ulceration of the cornea or nephritis sometimes complicates or follows the disease.

Diagnosis.—In well-developed cases the tenesmus, tormina, characteristic stools, and other symptoms render the diagnosis easy. Even cases which would otherwise be doubtful can be diagnosed if they occur during an epidemic, but isolated and atypical cases are often very difficult of recognition.

Syphilis and cancer of the rectum may produce bloody and mucoid stools with straining, but the course of the case, the absence of other dysenteric symptoms, and, frequently, rectal examination remove any difficulty in diagnosis.

Diarrhœa may simulate mild cases of dysentery, but differs in the absence of tenesmus and of mucous and bloody passages.

Some of the cases of acute diphtheritic dysentery, coming on with symptoms of great intensity, may strongly resemble typhoid fever. They are to be distinguished by the absence of enlarged spleen, rose-spots, bronchitis, and epistaxis, and by the presence of higher fever, severer intestinal symptoms, and the appearance at times of mucus and blood in the passages.

Cholera nostras and Asiatic cholera can be like dysentery only in the terminal algid state which is sometimes seen in the latter affection.

Intussusception in children may resemble dysentery in the presence of mucous and bloody stools, vomiting, colicky pain, tenesmus, and exhaustion, and it may be impossible to distinguish between them. Intussusception, however, is liable to exhibit a sausage-shaped tumor in the abdomen, and the fever is more obstinate.

Prognosis and Course.—The prognosis of dysentery varies greatly at different times and in different circumstances. Sporadic cases in temperate climates usually recover. On the other hand, some epidemics have been attended by a dreadful mortality, which may reach as high as 60 or 80 per cent. A more usual mortality, under favorable circumstances, is from 5 to 10 per cent.

The age of the patient influences the prognosis, old persons and infants suffering more than any other class. This is, however, not an invariable rule, since infants sometimes bear the disease very well. Epidemics developing in war prisons or among soldiers exhausted by long marches or insufficient food are prone to be exceedingly fatal. The occurrence of severe complications also increases the gravity of the prognosis.

The duration of the disease in the milder cases is eight or ten days from the onset of the distinctly dysenteric symptoms, but it may equal as much as three weeks. In the epidemic cases the duration is very variable. In the most malignant forms death may ensue in a few hours. In other instances the disease may last three or four weeks before a fatal issue takes place or convalescence begins.

Dysentery due to infection by the amœba coli runs an irregular course, with frequent periods of temporary improvement, during which the patient is about and fairly well. Its duration is from six to twelve weeks. The mortality is greater than in the sporadic catarrhal cases, and involvement of the liver is very likely to occur.

The course of chronic dysentery is very much prolonged, the disease lasting several months or possibly even years. The prognosis of this variety is most unfavorable, the patient often becoming extremely emaciated and dying of asthenia.

Asthenia is, indeed, the cause of death in most fatal cases of dysentery, except in those in which this results from some complications.

Relapse is uncommon in dysentery, and recurrence of the disease very unusual. The fact that the patient has once suffered from dysentery seems to offer a very great degree of immunity against subsequent attacks.

Treatment.—Treatment should be in the first place prophylactic. Overcrowding in cities, filth in dwellings, impurities in the water-supply, and improper quality of the food taken must all be guarded against. Every care must be observed to make the hygiene of army life and of prisons as perfect as possible.

If the affection has broken out, either epidemically or sporadically, precautions must be taken against its dissemination. With the possibility in view of the disease being contagious, it should be treated largely as though it certainly were so. Cleanliness of everything about the patient is to be enforced. Water drunk by others should be previously boiled, and the dejecta of the patient should be thoroughly disinfected. Those still in health should carefully avoid exposing themselves too much to the sun and to sudden changes of temperature, and should eschew unripe fruit and other indigestible food.

Various methods have been recommended for the treatment of the attack of dysentery. Perfect rest in bed is very important, and the use of the bed-pan should be insisted upon. The diet should preferably be of boiled milk only, or of whey, broths, egg-albumen, beef-juice, and the like. During convalescence great care must be taken that the food is of the most digestible kind.

If the case be seen early, it is well to aid in the evacuation of feculent matter by the administration of a saline purgative. Sufficient of it should be given to produce free passages, and it should then be discontinued. If scybala continue to be passed, it may be necessary to repeat the administration of the saline. In mild cases it sometimes happens that the dysenteric passages do not return and the tormina and tenesmus cease after this treatment has been used. If the stomach be irritable and the tongue heavily coated, fractional doses of calomel, gr. $\frac{1}{10}$, with bismuth, gr. iij, may be given every hour for ten or twelve doses.

In epidemic cases, or in any case where there is much prostration or where there are sero-sanguinolent stools, purgatives should be given cautiously if at all.

In these—and, in fact, in all cases—irrigation of the intestine by enemata may fittingly take the place of purgatives. The bowel may be thoroughly washed out with tepid water introduced through a long tube. The irritability of the bowel is so great that this procedure is often much interfered with. In such cases a cocaine suppository or a small injection of a solution of cocaine may be used as a preliminary step. The absorptive power of the rectum as regards cocaine must, however, be borne in mind, lest poisoning result.

Whether irrigation be used or not, the bowel should be treated locally by medicated enemata. Care is needed to adjust the size, strength, and frequency of these to the tolerance of the bowel, but the greatest good is to be hoped for from their judicious use. I advise beginning with small injections, f℥j or f℥ij, of a weak solution of silver nitrate, gr. $\frac{1}{2}$ or gr. j to f℥j, or of acetate of lead, gr. j to gr. ij to f℥j, guarded with from 5 to 10 drops of deodorized laudanum. If even these cannot be borne, small injections of ice-water or of starch-water may do better; or a small oblong piece of ice may be inserted into the rectum. If enemata are tolerated, but the small and weak ones fail to control the symptoms, larger and stronger ones should be used. Large enemata of a solution of nitrate of silver (1 drachm to 1 pint) have been remarkably successful in some instances. Alum (1 drachm to a pint), acetate of lead, sulphate of copper, and sulphate of zinc may be employed in a similar way. They are particularly useful in chronic dysentery. Enemata of salicylic acid have also been used with good results.

In amœbic dysentery injections of quinine (1 to 1000 and weaker) may be used with success.

Ipecacuanha has long been used as a specific against dysentery, and has without doubt been of very great service. It appears to be of more value in cases in the tropics than in those in temperate climates. It should be administered in large doses. It is best to give first a full dose of opium or morphine in order to aid the retention of the ipecacuanha, and then, after half an hour, to administer 20 to 40 grains of the remedy. No food should be taken for several hours after this. If the drug be vomited, the dose should be repeated within a few hours, and then should be given in diminished quantities at intervals during several days.

The severe tormina and tenesmus and profuse sero-sanguinolent discharges demand the use of opium, which may sometimes be given in enormous doses in this disease. The remedy may be administered by the mouth or, more advantageously in a great majority of cases, by the rectum or hypodermically, in sufficient amount to produce the effect desired, stopping short of narcotism.

Nitrate of silver is of decided value, though theoretically it might seem improbable that it would affect lesions so distant. It is best given in pill form in combination with opium, and has the added advantage that it is usually well received by the stomach. Among other remedies which have been advised are bichloride of mercury in doses of $\frac{1}{100}$ of a grain every two hours, bismuth in large and repeated doses, especially in chronic dysentery, ergotin, and various astringents given by the mouth. In cases which have passed into a fully-developed typhoid stage, a delicately prepared emulsion of oil of turpentine may be found beneficial. Turpentine stupes and hot fomentations to the abdomen often give relief, and the application of ice to the anus aids the patient in restraining the frequent efforts at defecation. Stimulants given freely are indicated in proportion to the degree of asthenia. The complications of dysentery require treatment appropriate for them.

Cancer of the Intestine.

Definition.—A morbid growth of the intestine, composed of cells of the epithelioid type, occupying alveoli of a connective-tissue stroma.

Synonyms.—Similar names are applied to the different varieties of intestinal carcinoma as are used for the forms of gastric cancer.

Etiology.—The disease is much less frequent than is cancer of the stomach. Primary cancer of the intestine has been estimated to constitute from 4 to 8 per cent. of all cases of the disease.

Cancer of the intestine occurs usually in the middle period of life and attacks either sex. It is questionable whether heredity exerts any influence. Constant irritation may act as a predisposing cause. Apart from these factors we are entirely ignorant of the etiology of the affection.

Pathology.—The varieties of cancer found as primary growths of the intestine are the same as those occurring in the stomach—viz. scirrhous, encephaloid, colloid, and cylindrical-celled epithelioma. Their appearance, structure, and method of growth are likewise the same as are presented in the gastric cancers. The cylindrical-celled epithelioma is probably the most common, although the colloid is also a frequent variety, and the scirrhous is often present in the rectum. The large intestine is affected by the growth about nine or ten times as often as is the small intestine, and the rectum about four times as often as all other parts of the bowel combined. Next to the rectum the favorite seat of cancer is the sigmoid flexure. The cæcum is also a point of predilection. In the case of the small intestine cancer of the duodenum is relatively common, and that of the jejunum rare.

Cancers begin in the mucous membrane and soon spread in depth and in area. There is a disposition shown by all the varieties to involve the whole of the intestinal wall, producing an annular growth, and in this way causing intestinal obstruction. Sometimes they are soft and fungoid, grow rapidly, crumble easily, soon ulcerate, and exhibit masses which project far into the lumen of the bowel. This is the case with encephaloid and cylindrical-celled epithelioma. In other instances, as in scirrhus, growth is slow and produces a very hard, cancerous infiltration. This form, too, ulcerates. Colloid cancer produces an alveolar structure of connective tissue, with the alveoli filled with a peculiar jelly-like yellowish substance. This variety of cancer is not so prone to ulceration.

The secondary lesions of the intestine produced by an obstructing cancer are best considered while discussing Intestinal Obstruction.

The cancerous growth often spreads to contiguous parts. Perforation into surrounding viscera or into the pelvic or peritoneal cavity may occur. Fæcal abscesses may form in this way. Sometimes a fæcal fistula through the abdominal walls is produced. Ulceration may be the cause of severe or fatal intestinal hæmorrhage or portions of the sloughing cancer may be passed in the stools.

All that has been said refers to primary cancer of the intestine. Secondary growths are more rare, yet the intestine may occasionally be invaded by cancer of other parts, particularly of the peritoneum, stomach, and lymphatic glands, and in the case of the rectum, of the uterus, bladder, prostate gland, and ovaries. Melanotic cancer may occur by metastasis from the skin or eye.

Symptomatology.—The symptoms of cancer of the intestine are entirely uncharacteristic. In the early stages there will be none at all. Later there is pain in the abdomen, usually localized in some special part. Emaciation and cachexia develop, and the bowels are irregular and often constipated, although sometimes there is diarrhœa. In other cases intestinal obstruction may be the first symptom noted.

When the growth is in the duodenum, evidences of obstruction of the pancreatic and bile-ducts may be present, and dilatation of the stomach may also result. Constipation and fæcal accumulation are more marked when the cancer is in the lower part of the bowel. Intense pain in the sacral region, radiating toward the genital organs and following the course of the sciatic nerve, may be present if the growth be in the lower portion of the large intestine. Cancer in the region of the sigmoid flexure is accompanied at times by frequent small fluid passages, which are sometimes very offensive, and which may contain blood, pus, and putrid masses.

Sometimes the stools in cancer are in the form of ribbon-shaped bands or in small round balls resembling the fæces of sheep. This is especially likely to occur in cancer of the rectum, but is not a positive indication of this lesion.

Rectal cancer usually produces intense pain in defecation. The bowels may be obstinately constipated, or there may be an almost continuous and uncontrollable evacuation of a thin, very offensive fluid.

Sometimes persistent constipation yields suddenly. This is an indication of the ulcerating away of the obstructing portion of the growth.

In the majority of cases a tumor can be detected by palpation of the abdominal walls. This may be movable or may be fixed by adhesions or by the nature of its location. If the growth be in the small intestine or colon, it may be remarkably movable. A tumor of the duodenum may not be distinguishable by palpation from cancer of the pylorus.

The cancer is usually tender on pressure, cannot be indented by the finger, may attain the size of the fist or more, and is irregularly round and uneven. It often seems to vary in size from day to day. So, too, it may be easily detected upon one occasion and on another be not perceptible at all. Percussion over the tumor produces a dull, tympanitic sound. The finger in the rectum can often perceive a growth situated in this region. A smooth annular constriction can be felt, or an irregular ulcerated surface which coats the examining finger with an ill-smelling, bloody fluid. Examination may also be made *per vaginam.*

Diagnosis.—The diagnosis of intestinal cancer is often difficult and sometimes impossible. When the tumor can be felt in the rectum the nature of the disease is evident. In place of being a growth of the intestine, a tumor felt through the abdominal walls may be a collection of fæces, a localized peritoneal exudation, an intussusception, or a growth of the peritoneum, liver, stomach, pancreas, omentum, kidney, or lymphatic glands. An accumulation of fæces can often be indented by the finger through the abdominal walls.

The diagnosis of the location of the affection is also difficult, since the weight of the tumor or adhesions formed may cause great deviations from the normal position of the intestine. The difficulties connected with the diagnosis should lead to great caution in expressing a positive opinion as to the cancerous character of a lump detected through the abdominal walls. Great suffering may be needlessly inflicted by the abrupt announcement of an unfavorable diagnosis which the subsequent course of the case proves erroneous.

Prognosis and Course.—The prognosis of intestinal cancer is absolutely unfavorable. The disease lasts at the most four or five years, and is usually of very much shorter duration. As the case nears the fatal issue marantic œdema is liable to develop, exhaustion grows extreme, and the patient dies from this.

Sometimes marantic thrombi form, and pulmonary emboli, resulting from these, are causes of death. In other cases a metastasis in other organs is the cause, and in still others intestinal obstruction, perforation, or fæcal abscesses with pyæmia kill the patient.

Treatment.—In some cases resection of the intestine has been of at least temporary benefit, and in others the formation of an artificial anus has prolonged life. In most instances, however, the treatment is purely medicinal and only palliative. A nourishing diet should be ordered, consisting of articles which form very little waste in the intestine, and nutrient enemata may be demanded in some instances. Mild laxatives are required from time to time. Morphine will usually be needed to relieve pain. Antiseptic and disinfectant enemata should be given several times a day if there be an offensive rectal discharge.

NON-CANCEROUS GROWTHS OF THE INTESTINE.

Most of these are so unusual that they possess only pathological interest.

Polypi may occur, especially in the rectum. They may be either pediculated fibromata or mucous polypi. They are commonest in children, and are liable to cause diarrhœa with loss of sufficient blood to produce marked anæmia. They sometimes project from the anus during defecation.

Lipomata are rare. Myomata, sarcomata, cystomata, and angiomata have been described. Lymphomata are not unusual.

ULCERATION OF THE INTESTINE.

For the sake of convenience of study a synopsis of the subject of intestinal ulceration may be of advantage:

1. *Round ulcer of the duodenum* is entirely similar to the round gastric ulcer, both in cause, pathology, and pathological anatomy, but is a much rarer affection. The situation of the ulcer is nearly always in the part of the duodenum above the entrance of the bile-duct. Below this the acid reaction of

the gastric secretion becomes neutralized, and one of the most important causes of ulcer is thus removed.

Duodenal ulcer is usually single, but is occasionally multiple. In some cases it occupies partly the duodenum and partly the pylorus. In other instances distinct ulcers may be found in the stomach and duodenum. The disease may attend septicæmia, probably as the result of embolism. Severe burns are also at times productive of it, and freezing is said to act in the same way, while erysipelas, pemphigus, and amyloid degeneration of the blood-vessels of the intestine have also been claimed to be associated with it. It is more common in males than in females.

The symptoms are in a great majority of cases identical with those of gastric ulcer, and it is then impossible to distinguish between the two lesions. An ulcer of the duodenum is probably present if the pain does not develop until some hours after eating food, if the position of this pain, together with sensitiveness on pressure, is situated decidedly to the right of the parasternal line, and if there are profuse bloody stools without hæmatemesis or vomiting of any kind. In other cases blood may both be vomited and passed by stool. In still others the course of the disease is entirely latent.

Ulcers of the duodenum show a particular tendency to fatal hæmorrhage and to perforation. The prognosis and therapy are the same as in the case of gastric ulcer.

2. *Follicular ulcers* have already been described as lesions developing frequently in the diarrhœal diseases of children, in chronic enteritis in the adult, and in dysentery.

3. *Catarrhal ulcers* have likewise already been mentioned as occurring under the same conditions as follicular ulceration. In the catarrhal form the ulcers are not situated in the lymphatic follicles, but in the mucous membrane proper.

4. *Stercoral ulcers* may result from the irritation accompanying prolonged retention of hardened fæces. This may be the result of active intestinal obstruction, may occur in the paralytic condition often present in old age, or may follow prolonged pressure of hardened masses which have lodged in the sacculi of the colon. Analogous to these latter is the ulcer occurring in the vermiform appendix. Stercoral ulcers are most frequent here and in the cæcum and sigmoid flexure.

5. *Lesions starting from without the intestine* may give rise to ulcers. Such, for instance, are cases of perforation of the intestine due to localized peritonitis dependent upon diseases of the adjacent organs or upon tuberculosis. An ulcer of this kind may also be produced by the erosion by morbid growths external to the bowel.

6. *Dysentery* produces more or less severe ulceration of both the follicular and catarrhal form. The disease is described elsewhere.

7. The *ulcers of typhoid fever* have already been described.

8. *Tuberculous ulceration* of the intestine is of frequent occurrence in advanced cases of pulmonary tuberculosis. It perhaps results from the

swallowing of tubercular sputum. The ulcers are seated chiefly in the solitary glands and Peyer's patches in the lower part of ileum, but may develop independently of these structures and in other parts of both the small and large bowel. The vermiform appendix is frequently attacked.

The ulcer is irregular in shape, with its greatest breadth usually horizontal to the long axis of the intestine, and with a tendency to follow the course of the blood-vessels which girdle the bowel. Sometimes it nearly encircles the gut. It penetrates into the submucous tissue and often into the muscular layer.

The edges and base of the ulcer are thickened and contain fresh and caseous tubercles, and in the vicinity of the lesion fresh tubercles can be found, which frequently form patches in the serous layer plainly visible to the naked eye.

9. *Syphilitic ulceration* is not common. It occurs in both the large and small intestine, and is generally the result of the breaking down of gummata. The ulcers are transverse or annular, and possess thickened, hard, grayish-white edges and an indurated, fibrous base, both of them densely infiltrated with round cells and often exhibiting cheesy nodules. Sometimes the primary syphilitic ulcer is produced in the rectum by pederastic practices.

10. *Cancerous ulceration*, or that from other morbid growths, may be produced by the breaking down of neoplasms of the bowel, as already stated.

11. *Toxic ulceration* of the intestine may occur. This is sometimes seen in poisoning by mercury or in any corrosive poisoning.

12. *Trauma* may produce ulceration, as in the case of that resulting from foreign bodies.

13. *Mycotic ulceration* is illustrated by the occurrence of ulcers in variola, anthrax, and actinomycosis.

The symptoms of intestinal ulceration naturally vary, and are to a large extent masked by those of the primary disease. If the ulceration be extensive, and especially if it be situated in the large intestine, there may be abdominal tenderness in the region of the ulcer. Appetite is often lost, though sometimes increased in cases of tubercular ulceration in children. Pain is liable to be produced, nutrition is impaired, and various evidences of indigestion may develop.

The character of the stools is important. Diarrhœa is likely to occur in the case of lesions of the large intestine even if there are but few ulcers here. On the other hand, extensive ulceration of the small intestine may be unattended by diarrhœa, and constipation may be present. Blood in the stools is an important symptom, although it may occur without ulceration, as when due to hæmorrhoids. It is particularly liable to be present in the case of duodenal ulcer. Pus in the stools is a strong indication of ulceration, although it may come from an abscess which has perforated into the bowel. Portions of tissue are found in the evacuations in the case of severe ulceration, and tubercle bacilli may be discovered when the lesions are of a tubercular nature.

Perforation with general or localized peritonitis or with fistulous openings may result from intestinal ulceration. Cicatrization may occur with constriction of the lumen of the bowel, this being a change particularly liable to follow syphilitic ulceration. Cicatricial stenosis follows tuberculous ulceration but rarely.

In many cases ulcers are found post-mortem which produced no symptoms during life.

INTESTINAL OBSTRUCTION.

Definition.—Mechanical interference with the passage of fæces through the bowel.

SYNONYMS.—Obstipation ; Ileus ; Entero-stenosis.

Etiology and Pathology.—Intestinal obstruction is a condition rather than itself a disease, since it is a result of several very distinct independent disorders. It may be due to structural disease of the intestine or to causes other than this.

The condition may also be acute or chronic, and may further be classified as partial or complete. Still, again, may be distinguished the cases in which stoppage of the circulation of the blood in the bowel occurs, and those in which there is simply obstruction to the passage of the intestinal contents, without any cutting off of the blood-supply.

Among the structural changes are to be included intussusception, internal strangulation or constriction, volvulus, stricture or compression, congenital malformations, and external hernia. The latter, although a cause of obstruction, cannot be considered in this connection.

Among causes other than structural are functional obstruction and impaction of fæces or of foreign bodies.

INTUSSUSCEPTION.—This disease, also called *invagination*, is a frequent cause of intestinal obstruction. It is variously estimated to form 25 to 45 per cent. of all cases of obstruction. The greatest frequency is, however, in children, in whom fully one-half of the cases occur. In adults the disease is not as common as the other forms of obstruction.

The condition is not infrequently found after death, especially in infants, without there having been any symptoms during life. Such invaginations can be very easily reduced, and there is no evidence of inflammation about them. They have probably arisen at the moment of death, and are entirely different from the invagination occurring during life, which is attended by evidences of inflammation with symptoms of intestinal obstruction. It is with this latter condition alone that we have to do.

The active cause of invagination is irregular peristalsis—probably a paralysis of a portion of the intestine with increased peristalsis of an adjacent portion. Colic, diarrhœa, and constipation seem to favor the development of the lesion, but in fully one-half of the cases the accident occurs without the slightest warning.

The invagination forms a cylindrical tumor which varies greatly in length and may include from a few inches up to several feet of intestine. One portion of the intestine slips into an adjoining part. In this way three layers of bowel are brought into apposition—the external layer, called the *intussuscipiens* or *receiving* layer, and the middle or *returning* layer, in contact by their mucous surfaces; the middle and inner or *entering* layer, together called the *intussusceptum*, in contact by their serous surfaces.

The invagination is almost invariably downward, the portion above entering that which is below.

There are several varieties of intussusception, according to the portion of the bowel affected. Much the most frequent of these is the ileo-cæcal variety, in which the ileo-cæcal valve slips into the large intestine, gradually inverts this, and descends even into the rectum, dragging the small intestine after it. The valve can sometimes be felt at the anus. In another form the ileum slips through the ileo-cæcal valve, and in still others the small intestine or the colon alone is involved.

Inflammation of the invaginated portion of the bowel produces adhesion of the apposed serous surfaces with consequent incarceration. The circulation of the blood is interfered with by the tension and constriction of the mesentery, and congestion and swelling follow and produce obstruction of the lumen of the intestine. The affected part of the bowel becomes livid and ecchymotic. If the disease lasts two or three days, more or less extensive peritonitis is likely to develop around the incarcerated bowel, and so much lymph is thrown out that a reduction of the invagination is impossible without tearing the bowel. If the constriction of the vessels has been very great or prolonged, gangrene of the bowel develops, and the invaginated portion may slough away if the patient live long enough.

If death occur within the first twenty-four hours, very little abnormal can be seen on post-mortem examination, except some congestion of the invaginated bowel with delicate adhesions between the serous surfaces.

Sometimes the extreme engorgement of blood in the bowel does not occur, and the intussusception becomes chronic, and may last for months or even, very rarely, for one or two years. In these cases the lesion is generally ileo-cæcal or colic. Pain is paroxysmal, with periods in which it is entirely absent. Constipation is not complete, and vomiting is not constant. After a time mucoid and bloody evacuations from the bowel develop, pain and vomiting grow more troublesome, and the patient finally dies from exhaustion.

INTERNAL STRANGULATION.—This disease, also called *Constriction of the bowel* or *Hernia within the abdomen*, is one of the most frequent causes of intestinal obstruction, equalling about 35 per cent. of all cases. In adults it is probably the most frequent cause. It is estimated that 40 per cent. of all cases occur between fifteen and forty years of age, and 70 per cent. in males. The small intestine is the portion by far most frequently involved.

The strangulation may be produced in various ways. Fibrous bands and adhesions resulting from peritonitis are very common causes. Strangulation may be produced by a loop being held down by such a band or by being twisted around it.

A not infrequent cause of constriction is an abnormal attachment of the free end of Meckel's diverticulum to the mesentery or to the abdominal wall, thus forming a band which may strangulate some portion of the bowel. The adherence of the tip of the vermiform appendix to some adjacent part may act in a similar manner, but is a much rarer occurrence.

Also only rarely seen are strangulations due to constriction of the bowels in holes in the omentum or mesentery or in the normal peritoneal openings or pouches—such, for instance, as the foramen of Winslow and the duodeno-jejunal fossa—and hernia through the diaphragm is not an infrequent cause of internal strangulation.

The ileum is the portion of intestine involved in nearly all cases of strangulation. The accident produces intestinal obstruction, with ulceration and sometimes even sloughing of the strangulated portion, and consequent perforation.

VOLVULUS.—Volvulus, or a twist or knot of the bowel, is an occasional cause of intestinal obstruction. It forms from about 3 to 10 per cent. of all fatal cases. It is rare in children.

The lesion nearly always consists in a twist in the long axis of the intestine. In other cases one loop of bowel is twisted around another or the gut is bent sharply upon itself.

The commonest situation is the sigmoid flexure. The cæcum is also a frequent seat of the accident. The twisting of the intestine produces interference with the circulation of blood and its natural results, together with complete fæcal obstruction.

STRICTURE AND COMPRESSION.—Narrowing of the intestine may be produced either by a stricture situated in the intestinal wall or by compression from without. Acute obstruction rarely follows from such a cause, but chronic obstruction is frequently produced in this way. Interference with the blood-supply does not develop in this form of intestinal obstruction, as it does in the conditions previously described. In the large majority of cases the diminution of calibre is situated in the rectum or the sigmoid flexure. It is generally not a complete occlusion, but some fæcal matter still passes.

Of the various causes of narrowing, congenital stricture is a rare one. Its situation is nearly always in the rectum. When elsewhere, it is usually in the sigmoid flexure or in the lower portion of the ileum and at the ileo-cæcal opening. The condition is the result of fœtal peritonitis.

More common is some congenital malformation, such as imperforate anus or imperforate rectum, with complete occlusion of the bowel.

Cicatricial contraction following ulceration is one of the most common causes of stricture. This may be the result of dysenteric, syphilitic, stercoral, tubercular, or other ulceration. Sometimes cicatricial constriction may follow sloughing of the invaginated portion of the intestine, or the ulceration produced by an irritant poison or by the presence of foreign bodies.

Tumors in the intestinal wall, whether malignant or benign, are very frequent causes of stricture. Cancer is the most common of these. It is much most frequently seated in the rectum, where it forms an annular growth around the bowel, encroaching upon the calibre of the tube and often narrowing it to an extreme degree. Sometimes masses of the growth ulcerate away, and improvement in the symptoms of obstruction follows.

Narrowing of the lumen of the intestine may also be produced by compression from without. Thus tumors or cysts of neighboring organs may exert pressure or traction upon the bowel and produce intestinal obstruction. This is especially true of growths of the pelvic organs. Sometimes, though less often, a loop of intestine distended with fæcal matter compresses some adjacent loop, or a contracting inflammatory exudate, resulting especially from tubercular peritonitis, produces partial or, more seldom, complete occlusion of intestine. The mutual adhesion of coils of intestine and their compression in this way by the peritoneal inflammatory tissue is a common cause of intestinal obstruction.

Functional Obstruction.—This disease, also called idiopathic ileus, is one of the more unusual causes of non-structural obstruction of the bowel. It is an instance of acute obstruction without interference with the circulation. It occurs chiefly in hysterical or nervous individuals, particularly women, but may also result from diseases of the brain and spinal cord, from peritonitis, or from blows upon the abdomen. It may also follow the reduction of a hernia. It is the result of the paralysis of a portion of the intestine, in consequence of which peristaltic movements in this region cease and the fæces no longer advance.

Impaction of Fæces and of Foreign Bodies.—This is not an infrequent cause of an obstruction which is usually of a subacute or chronic nature. Impacted fæces are, as a rule, situated in the colon or rectum. The contents of the bowel may become very hard, and may form a tumor which is easily appreciable through the abdominal walls, and which may even be of great size. The mass is often channelled in such a way that small amounts of fæces can pass, thus rendering the obstruction only partial. Finally the channel becomes blocked and the occlusion is complete. Impaction of fæces may occur at any age, but is oftenest seen in those past middle life.

Gall-stones not uncommonly become impacted and produce obstruction. They are found almost invariably in the small intestine, and oftenest in the ileum very shortly above the ileo-cæcal valve. The impaction of gall-stones has usually occurred in advanced middle life, and most frequently in women. One large stone or a cohesive mass of smaller ones usually enters the intestine by ulceration through the gall-bladder and intestinal wall.

Enteroliths, although frequent in herbivorous animals, are a rare cause of obstruction in man. They are generally composed chiefly of phosphate of lime and magnesia deposited around a central nucleus, which consists of a gall-stone, a mass of hardened fæces, or some foreign body. Enteroliths sometimes occur in persons who subsist largely upon oatmeal. The nucleus then consists of portions of oats. The bodies have then been called "avenoliths." Starch or other vegetable substances may produce similar concretions. Enteroliths are generally found in the cæcum or the sacculi of the colon, and rarely in man exceed the size of a hazel nut.

Foreign bodies—*i. e.* substances not a portion of the food or arising from the human body itself—are sometimes the cause of intestinal obstruction. Masses of intestinal worms have rarely caused acute obstruction. Foreign bodies which have been swallowed by accident or design, especially by children or lunatics, may produce obstruction, particularly if they are sharp-pointed or of irregular shape. The great majority of all these bodies, however, are passed *per anum* without any symptoms developing.

Allied to enteroliths are the dense masses which may come from accumulation in the bowel of magnesia, bismuth, iron, or other insoluble substances taken for a long period as medicine.

Of the causes of obstruction mentioned, intussusception, internal strangulation, volvulus, impaction of foreign bodies, gall-stones or enteroliths, congenital malformations, and functional obstruction usually produce symptoms of acute intestinal obstruction, while impaction of fæces, stricture and compression generally produce chronic obstruction. Impaction of fæces especially is liable to develop chronically, but suddenly at last to produce the symptoms of acute obstruction.

Intussusception, internal strangulation, and volvulus not only cause obstruction, but are attended by interference with the circulation of blood in the bowel and the consequent danger of rapid death of the part.

In addition to the pathological changes described as occurring at the seat of obstruction, certain secondary lesions are to be noted. The portion of the intestine above it is necessarily dilated, owing to the accumulation of fæces and of gas. Especially in the chronic cases is the amount of fæces present very great. In the acute cases the distended intestinal wall is thin and transparent, while in the chronic ones, it is thickened and the muscular layers are hypertrophied. In any case the distention may at last become so great that rupture ensues. The mucous membrane of the intestine above the obstruction may ulcerate or become necrotic in patches as the result of mechanical irritation by scybalæ.

Changes may be produced in other organs, especially the lungs, in which inspiration-pneumonia is liable to develop, as the result of the passing of vomited matter into the larynx and deeper. The tissues in general often appear exceedingly dry at the autopsy, this being the result of the abstraction of water by the excessive vomiting.

Symptomatology.—The symptoms of occlusion of the intestine must be divided into those of the acute and those of the chronic form.

1. ACUTE OBSTRUCTION.—The first symptoms often develop in the midst of apparently perfect health. In a smaller number of cases, however, there are various premonitory digestive disturbances, such as moderate diarrhœa, constipation, or slight abdominal pain. The characteristic symptoms of the disease are pain, vomiting, and constipation.

Extreme pain in the abdomen sets in suddenly, even while the patient is walking about. It is at first localized at some part of the abdomen, and is colicky and intermittent, but later it becomes continuous, although usually with exacerbations, and is often agonizing. Although it may finally spread all over the abdomen, it still, as a rule, continues most intense at the point at which it was at first experienced.

Vomiting very soon develops, at first of food, then bilious in character, and then, after two or three days, stercoraceous if the obstruction be complete. The vomiting is incessant and most distressing. Stercoraceous vomiting may occur whether the obstruction is in the large or small intestine. If the occlusion be prolonged, fæcal vomiting may cease and the ejected matter may consist of a rice-water-like fluid. Sometimes toward the close of life vomiting may be entirely replaced by most distressing hiccough.

Constipation soon becomes absolute in case of acute complete obstruction, although at first there may be the passage of fæcal matter, which was still present in the lower part of the intestine when the obstruction occurred. There is also an entire absence of the passage of flatus. In intussusception there are bloody mucous discharges in addition to entire retention of fæces.

Unless the obstruction is high up in the small intestine the abdomen soon becomes tympanitic, and when the occlusion is in the large intestine the distention may be extreme. Very active peristaltic movements can often be perceived in the intestine above the seat of obstruction, and these are accompanied by borborygmi so loud at times that they may be heard at a considerable distance.

The abdomen is at first not tender, but may later become very sensitive to touch. Palpation detects a tumor in some cases. Examination, *per rectum* and *per vaginam*, frequently reveals much regarding the position of the obstruction or the possible influence of disease of the pelvic organs in producing it.

The constitutional symptoms are severe from the beginning, and the patient may rapidly reach a very alarming condition. There is great restlessness, the face is pale and cold, the eyes sunken, the nose sharp and the features pinched, the voice whispering, and the skin cold and clammy. The pulse is small and either rapid or slow, and the temperature may be slightly elevated, but is often normal or subnormal. The tongue is dry, thirst greatly increased, the mind generally clear, and cramps may develop, as in cholera. The urine is very scanty, or even suppressed, as the result of the excessive vomiting and the large amount of fluid lost in this way.

2. Chronic Obstruction.—The symptoms in this condition develop only gradually. The patient has shown evidences of abnormal disorder perhaps for weeks or months; sometimes with a history of long-standing constipation which has not yielded satisfactorily to purgatives. Not infrequently the fæces assume a peculiarity, being ribbon-shaped or in the form of small round masses like the fæces of sheep. Sometimes they exhibit a furrow on one side. This alteration of the shape of the fæces is, however, not decisive, as it may occasionally appear in other conditions.

In many cases abdominal pain is present at times, although not so excessive as in acute obstruction. Vomiting is not a common symptom. The abdomen is often greatly distended, and the increased peristaltic movements of the intestine may be distinctly visible and be attended by very loud borborygmi, as in acute obstruction. Very often the masses of fæces which have accumulated can be felt through the abdominal wall. Rectal and vaginal examinations are even more important than in the acute disease.

The health of the patient gradually fails, and finally, when the occlusion becomes complete, the symptoms of acute obstruction may develop or the case may still run a more protracted course.

Diagnosis.—In the study of intestinal obstruction it is necessary to determine, if possible, the seat of the lesion. This is often a matter of the greatest difficulty. Rectal and vaginal examinations should always be practised. The former may reveal obstructions situated in the rectum or sometimes a descending invaginated bowel. Vaginal examinations show the condition of the pelvic organs, and their possible influence in producing obstruction of the intestine. In cases in which the obstruction is high in the small intestine the collapsed coils of bowel below the lesion sometimes sink into the pelvis and can be discovered there. Pain is often confined to the seat of obstruction, and palpation through the abdominal walls may reveal a tumor in this position. The situation of the tumor is not always, however, an indication of the portion of the bowel obstructed, since by its weight it may drag the affected intestine out of its normal place.

If the obstruction be in the duodenum or jejunum, vomiting occurs early, the abdomen is not distended, the urine is probably entirely suppressed, and collapse is rapid.

If the ileum or cæcum be the portion obstructed, the umbilical region of the abdomen is especially distended, and the belly globular in shape, with the flanks flattened. The course of the case is very rapid, and vomiting is early and often fæcal.

If the obstruction be low in the colon or in the rectum, the abdomen is distended all over, including the flanks. There may or may not be tenesmus. The symptoms are not so intense, and the amount of urine is not so much reduced, as when the small intestine is affected. The recognition of the seat of an obstruction in the large intestine is sometimes aided by the quantity of fluid which can be injected into the bowel. Normally, the adult large intestine should hold about six quarts, but no obstruction can be asserted if four quarts can be introduced. The injection must be made carefully with a fountain syringe, the elevation of which above the patient should not be more than a few feet. If the endeavor thus to fill the colon and cæcum is successful, the obstruction must be in the small intestine. The method is, however, uncertain in its results. So also is that of the introduction of a rectal sound, since the instrument cannot be safely passed farther than the sigmoid flexure.

The symptoms as described are those which apply in general to cases of acute and chronic obstruction respectively. It is still necessary to consider briefly the distinguishing symptoms of some of the different causative conditions which produce the obstruction, although it is often difficult or impossible to determine what the nature of the occlusion is.

Intussusception is usually the easiest of recognition. It is essentially a disease of childhood, and is the cause of the obstruction of most cases in children. A tumor, which is oftenest situated in the right iliac fossa, can be detected by palpation in most of the cases as early as from the first to the third day. The presence of tenesmus with the passage of blood-stained mucus is a very important symptom which is present in a great many instances. Distention and tenderness of the abdomem is not an important symptom, and sometimes the abdomen is retracted. Vomiting occurs very early, but fæcal vomiting is not common. Prostration is early and profound. The finger in the rectum can sometimes reach the invaginated bowel.

In internal strangulation there is often a history of preceding peritonitis or abdominal injury, or at least of pelvic pain. The attack itself is sudden and liable to come on after exertion or indigestion. Pain is excessive, vomiting is early and copious and soon becomes fæcal, and the very extreme prostration is one of the most characteristic features. As a rule, no tumor can be felt. Constipation is absolute, and there is no tenesmus or passage of mucus or blood. Abdominal distention and tenderness do not occur until late in the disease. Rectal examination reveals nothing.

Volvulus cannot be recognized with certainty during life. The fact that it is nearly always situated at the sigmoid flexure is important. There are generally premonitory constipation and digestive disturbances, the twist being at last produced by the weight and tension of the accumulated fæces. The actual onset is very sudden and severe, constipation and retention of gas are absolute, and there is no tenesmus or passage of mucus or blood by the rectum. Vomiting comes on rather late, and only occasionally becomes fæcal. No tumor can be felt through the abdominal walls or by rectal examination. Distention of the abdomen with gas and abdominal tenderness are early and prominent symptoms. Prostration and collapse are not so great as in intussusception and strangulation.

Obstruction by a foreign body gives varying symptoms. There may be the history of the swallowing of some such body, and sometimes it can be felt through the abdominal wall. There may be for days abdominal pain and discomfort. If, finally, the body becomes firmly lodged, the intestine begins to swell and obstruction is rendered complete. Pain now increases gradually, and tenderness and distention of the abdomen rapidly develop, but vomiting is late in appearing, and is not constant.

In obstruction due to gall-stones the patient has in some cases recently suffered from an attack of hepatic colic or has on previous occasions done so. In one case, the interval between the close of a series of attacks of hepatic colic and the sudden occurrence of complete obstruction was two years, during which time a very large round gall-stone, which had ulcerated its way from the gall-bladder into the duodenum, had lain quiescent and attached to a portion of the wall of the dilated duodenum. Pain usually is an early and severe symptom. Vomiting likewise is early, and is very frequently fæcal. A tumor is not often to be detected. If the impaction be situated in the duodenum there is persistent bilious vomiting, with rapid collapse and retraction of the abdomen and great diminution or suppression of the urine.

In fæcal obstruction the onset is usually gradual, and the disease runs a chronic course, with habitual constipation and finally, perhaps complete, obstruction. Abdominal pain, nausea, and vomiting are not constant and are late in developing. In some cases fæcal retention lasts weeks without any threatening symptoms being produced, and the masses may be finally discharged. In other cases there develop abdominal pain, gradually increasing distention, and vomiting, which is finally stercoraceous. A fæcal mass can often be felt by rectal examination or by abdominal palpation over the course of the colon. This mass occasionally reaches great size. Diarrhœa sometimes replaces constipation, this being the result of a chronic catarrhal process set up by the hard fæcal masses. In such cases there exists a channel in the impacted mass through which the softer fæces pass.

Narrowing of the intestine by stricture or compression exhibits much the same mode of onset as does fæcal impaction. The symptoms of the condition vary. There is persistent chronic constipation, perhaps with attacks at times of temporary complete obstruction attended by vomiting, which is sometimes stercoraceous, and by abdominal distention and violent peristaltic movements. The passages are apt to be ribboned or grooved, defecation may be painful, and blood and pus may coat the stools if the stricture be in the rectum and of a cancerous nature. When the narrowing is due to compression from without, as by a tumor, blood and pus do not appear in the fæces. Even when the obstruction to the passage of the fæces becomes absolute the case may still run a comparatively slow course.

In congenital stricture and malformations the history of the case is sufficient to establish the diagnosis.

Functional obstruction cannot always be distinguished from acute structural affections. The absence of localized pain and tenderness and of tumor, and, in some cases, the evidence of hysteria or nervous symptoms, indicate the functional nature of the disturbance. So also in these latter cases does the prolongation of the threatening symptoms without the development of extreme prostration, feeble pulse, collapse, and other evidences of the destructive lesions of the bowel.

Not only are the forms of intestinal obstruction to be distinguished from each other, but the diagnosis is to be made between intestinal obstruction in general and other diseases. Acute obstruction is particularly liable to resemble hernia, and the mistake of confounding the two has been repeatedly made. A careful search for an undiscovered strangulated external hernia should be made in every case, such small hernias being very easily overlooked. The diagnosis, too, is particularly difficult in cases of intestinal obstruction complicated by chronic irreducible hernia, since, if the obstruction is in the bowel below the position of the rupture, the portion of the intestine in the sac will become swollen and hard from the accumulation of fæces, and will appear to be strangulated.

Functional obstruction, due to functional or organic nervous diseases, has already been spoken of, and the fact mentioned that this condition may also be a complication of peritonitis or may follow the reduction of a hernia. In the latter event the history of the case, and in the former the existence of such symptoms as fever and the more rapid development of widespread abdominal tenderness, distention, and pain, aid in distinguishing the primary diseases.

Appendicitis with secondary peritonitis may produce symptoms resembling mechanical obstruction. Vomiting is, however, rarely fæcal, the temperature is more often elevated, and there is frequently a history of previous trouble in the cæcal region.

Acute hæmorrhagic pancreatitis may be attended by symptoms so resembling those of intestinal obstruction that a diagnosis is scarcely possible.

Poisoning by irritant poisons, as by arsenic, may resemble intestinal obstruction in some particulars. The history of the case and the character of the vomited matter aid in the diagnosis.

Prognosis and Course.—The prognosis of every case of acute intestinal obstruction, with stoppage of the circulation of blood in the intestine, is most grave. The duration of life varies inversely with the height of the obstruction in the intestine and the amount of injury which the bowel has received. It ranges from a few hours to ten or twelve days, with an average of three to six days. The great proportion of cases of this nature die, death resulting from exhaustion if the patient live long enough. Sometimes death occurs very early from shock, while in other instances a vast accumulation of gas produces death by interference with respiration or circulation. Rupture of the intestine and perforative peritonitis may also occur, or peritonitis may develop without perforation.

The prognosis of volvulus and of internal strangulation is totally unfavorable, with the rarest exceptions, unless relieved by surgical measures. That of intussusception is perhaps a little better, since the development of gangrene may occasionally produce a favorable termination, the invaginated portion of the bowel sloughing off and being discharged *per anum.* This occurs from the eleventh to the twenty-first day, but, unfortunately, few children live long enough for it to take place. Even where it does occur stricture of the gut is liable to develop later.

In exceptional cases of intestinal obstruction recovery may occur by the formation of a fistula either between two coils of the intestine or through the abdominal walls. Often perforation of the bowel, which does not produce general peritonitis, causes death from pyæmia.

The prognosis of functional obstruction due to hysteria or neurasthenia is much more favorable, the obstruction usually yielding at last. In that due to peritonitis or reduced hernia it is dubious, but favorable as compared with that of intussusception, volvulus, and internal strangulation.

Acute obstruction from impaction of foreign bodies, gall-stones, or enteroliths may recover through the passage or removal of the obstructing bodies.

The prognosis of chronic obstruction, as in fæcal impaction, stricture or compression, is much less grave than in the acute form. Cases of fæcal impaction usually recover, while those of stricture or compression may last a long time, although they usually become worse gradually unless the cause be removed. Death from chronic obstruction may result from perforation, pyæmia, or the like, and it is still more liable to follow from asthenia or from the development of acute obstruction. The duration of chronic obstruction is exceedingly variable, the disease lasting months or even years.

Treatment.—Treatment, to be of any value, must be energetic and promptly carried out. If the obstruction be due to impaction of fæces, as large injections of warm water as can be administered should be given every two hours. Drastics are to be avoided, but mild purgatives may be given in small and repeated doses. This plan of treatment should not be followed if there are evidences of inflammation until these have been allayed. A delicately made emulsion of castor oil containing a little oil of turpentine given in small and frequently repeated doses has proved very useful. Abdominal massage may be of service. If the obstruction becomes complete and these remedies fail, surgical interference must be adopted.

In many cases of fæcal impaction treatment by opium is more successful than that by enemata or purgatives. After the relief by it of pain and vomiting, any element of spasm present is relaxed and evacuation of the bowels may follow of itself. This plan of treatment may well precede the use of enemata or purgatives. After the impaction has been relieved, it is necessary to guard against a recurrence by care in diet, proper gymnastic exercises, and the internal use of tonics and especially of strychnine to improve the tone of the bowel.

Cases of obstruction from stricture or compression require much the same treatment as do those due to fæcal impaction if the obstruction is not complete and there is a large amount of fæces above the stricture. Surgical interference is the only measure capable of giving permanent relief. Tumors pressing upon the bowel should be removed, and cancerous or other strictures operated upon if possible.

In all cases of chronic intestinal obstruction it is important to prevent the recurrence of acute attacks. For this purpose the chief care must be for the diet. The food should consist of articles which make but little waste, and these should be carefully masticated, so that no large, firm masses shall reach the narrowed portion of the intestine. If necessary, the fæces should be kept moderately soft by the use of a laxative mineral water or some mild vegetable cathartic.

It is of the greatest importance to remember that purgatives should be given in no case of intestinal obstruction, except in those due to impacted fæces and in those the result of stricture or compression, and that they are not always advisable even in these.

In cases of firmly-impacted foreign bodies, enteroliths, or gall-stones, in which the impaction does not promptly give way of itself, laparotomy offers the only hope of cure.

In cases of occlusion from internal strangulation, volvulus or intussusception the first step should consist in the administration of opium. Opium is, in fact, invaluable in nearly all cases of acute intestinal obstruction. Not only does it relieve excessive pain and vomiting, but it greatly lessens the danger of death from collapse. Then, too, by reducing the peristaltic movement it diminishes the danger of serious injury to the intestine, moderates inflammatory action, and gives the affected bowel a chance to untwist or to escape from compressing bands or from an invagination.

The next procedure which should be resorted to in all cases of complete obstruction before surgical interference is at all thought of is the repeated slow injection of large quantities of warm water or of air. This may best be done with the patient under the influence of an anæsthetic, and in the knee-elbow position if water be used. In intussusception this method is often successful. It should not be attempted, however, if the invagination is believed to have become firmly fixed,—*i. e.* after the case has lasted forty-eight to seventy-two hours or if gangrene has commenced. It is also a procedure by no means without danger, as rupture of the bowel may result if an undue amount of force is used.

It has also been recommended to treat these gravest forms of abdominal obstruction by abdominal massage while the patient is anæsthetized, by abdominal traction with large cups, or by shaking the patient thoroughly under anæsthesia and while held in the inverted position. Nothing is to be expected from any of these plans if inflammatory fixation has occurred.

Tapping the bowel with a hollow needle to permit the escape of gas and to give temporary relief has been advised. Sometimes relieving the distention in this way has permitted spontaneous reduction of an internal strangulation or invagination or the untwisting of a volvulus. The procedure has a large element of danger of peritonitis from escape of fæces. It is, however, of value in some cases. Turpentine stupes and hot applications are of service in milder cases of tympanites.

If none of these measures succeed within forty-eight hours, operative interference must be resorted to promptly if at all, for later than this the condition of the intestine is such that the chances of recovery are very greatly lessened. Various operations may be performed, such as the untwisting of a volvulus, the freeing of a strangulated bowel, or the reduction of an intussusception; or, in cases too advanced for these procedures, the formation of an artificial anus, resection of the bowel or lateral anastomosis. The consideration of these operations belongs to works on surgery.

Although statistics show a large proportion of deaths after operation for intestinal obstruction, yet there would be without surgical interference a still larger number of fatal cases. There should be no question about operating in such conditions as internal strangulation, volvulus, and impacted foreign bodies and gall-stones, in which death is otherwise inevitable. In cases of intussusception there is more doubt about the propriety of interference, because in this condition there is a somewhat greater chance for recovery without it, and the statistics of operation are very unfavorable. At the same time, there appears to be a much greater chance for life with operation than without it in cases which have resisted mechanical treatment and in which the operation is not deferred beyond the third day at the latest.

Throughout the whole course of the case of acute intestinal obstruction it is of great importance to support the strength of the patient by stimulants and by concentrated, easily-assimilable food. If, as often happens, vomiting is easily excited, it may be necessary to avoid entirely the administration of food, to allow only small sips of water, of carbonated water, of champagne or brandy and water, and to resort to nutritious enemata, to inunction, and to hypodermic injections of whisky or ether. It may be possible to sustain the strength of the patient until the obstruction is relieved.

Hæmorrhage from the Intestine.

Etiology.—Hæmorrhage from the intestine (Enterorrhagia; Enterohæmorrhagia) is a symptom which can be produced in various ways. When it proceeds from the intestine itself, it is enterorrhagia in the strict sense of the word. *Melæna* is a term very loosely applied. It oftenest indicates dark-colored or black passages from the bowel, consisting of blood which has remained long enough in the stomach or intestine to have become thus altered in appearance. Intestinal hæmorrhage occurs most frequently in middle life, and oftener in males than in females. It is rare in infants.

Obstinate constipation may produce hæmorrhage by direct injury of the intestinal wall. The bleeding in this case nearly always comes from the rectum and is slight. Often it coats or streaks hard fæcal masses evacuated.

Local diseases of the intestinal wall are also a source of hæmorrhage. *Intense hyperæmia* of the mucous membrane is a cause which may be included here. Such a hæmorrhage may occur in dysentery, even when no ulceration can be discovered. It may also be produced by obstruction of the portal vein or by disease of the lungs, liver, or heart. In some of these cases the blood does not come directly from the mucous membrane, but from dilated hæmorrhoidal veins. Embolism of the mesenteric artery may produce hæmorrhage by causing congestion and partial infarction of the mucous membrane. Intussusception likewise produces it through the intense obstructive hyperæmia of the mucous membrane.

Traumatism of the intestine by a foreign body of any sort may be a cause of hæmorrhage. Such trauma may follow the swallowing of some irregularly-shaped or sharply-pointed object. In the same category, though this cause is operative only in certain parts of the world, may be included hæmorrhage following a lesion produced by intestinal parasites—viz. the anchylostomum duodenale and the distomum hæmatobium. Poisonous drugs, including powerful drastic purgatives, occasionally produce hæmorrhage by their violent action upon the intestine.

Amyloid degeneration of the intestinal vessels has been found to be a frequent cause of bleeding from the bowel.

Ulceration is a very common cause of hæmorrhage. Bleeding may occur from any variety of ulcer, but typhoid ulceration is one of the forms which most frequently gives rise to it. The bleeding may be slight, or very often profuse and perhaps fatal. Dysenteric ulceration is very prone to attest its presence by blood in the stools, and the hæmorrhage may be fatal. Tubercular and syphilitic ulceration are less frequent causes of hæmorrhage. Ulcer of the duodenum very commonly is the cause of great loss of blood.

Morbid growths of the intestine may be attended by hæmorrhage. Here are to be included cancer and polypi. In the former blood is frequently lost, although usually in small amount. The latter form of growth is generally situated in the rectum, and is characterized by frequent and sometimes profuse loss of blood. Under the heading of new growths may be included internal hæmorrhoids, which are a very fruitful source of bleeding, sometimes slight, sometimes very profuse.

General diseases may occasion enterorrhagia without this proceeding from any ulceration. Hæmorrhage of this kind is seen occasionally in malaria, typhoid fever, and cholera. In yellow fever it is quite common. It also may occur in variola, uræmia, pyæmia, erysipelas, hæmophilia, pernicious anæmia, purpura hæmorrhagica, acute fatty degeneration of the newly-born, scurvy, and leukæmia. Vicarious menstruation may show itself by enterorrhagia.

Causes outside of the intestine itself may produce hæmorrhage, as when blood from the stomach enters the bowel, or when an aneurism ruptures into the intestine from without.

Melæna neonatorum is a term applied to vomiting and the passage from the bowel of discolored blood occurring a few hours after birth. Sometimes the loss of blood is sufficiently rapid to produce death. The causes of this condition are not well understood. In some instances no anatomical lesion or other source of hæmorrhage can be found except intense hyperæmia of the mucous membrane, while in others the cause appears to be ulceration of the stomach or duodenum, general dissolution of the blood within the vessels, hæmophilia, or puerperal infection.

Pathology.—The lesions found correspond to the causes mentioned, and are best described in connection with the various diseases to which the hæmorrhage is due.

Symptomatology.—Sometimes the hæmorrhage is entirely internal, no blood being voided. The patient then becomes very pale, and exhibits all the usual symptoms characteristic of sudden and great loss of blood. Death may occur without any blood appearing. In some of these cases a great increase of dulness over the abdomen is perceptible. The examination, however, must be cautiously made.

The usual chief symptom of intestinal hæmorrhage is the appearance of bloody stools. The blood may be pure or mixed with fæces or mucus, or may be altered in character. Often the passages are tarry in appearance and very offensive. In other cases they form hard, black masses. At times tenesmus accompanies the evacuation of the bowels, and colicky pain may occur.

Diagnosis.—In the first place, it is necessary to determine that it is blood which is being passed. There is generally no difficulty about this, except in the cases in which the blood has been much altered. Here the microscopic, chemical, or spectroscopic tests for blood will settle the matter. Sometimes the ingestion of certain raw red fruits or of preparations of hæmatoxylon produces a red coloration of the stools.

That the blood actually is effused in the intestine and does not enter it from elsewhere can only be determined by carefully excluding all such conditions as gastric ulcer, swallowing of blood after epistaxis or operations on the mouth, or, in the case of infants, the drawing of blood from a sore nipple.

The diagnosis between bleeding from a gastric ulcer and that from duodenal ulcer is often impossible. The points of distinction have already been referred to elsewhere.

Effort must be made to determine the situation whence the blood comes. Physical examination of the abdomen and, especially, of the rectum, and the consideration of the probable cause of the hæmorrhage, will also aid in settling this point. The color of the blood is often important. If it be bright red and small in amount, it comes almost certainly from the large intestine. This is also the situation if the blood is smeared over formed fæcal masses. If the blood is in large quantity, yet much altered in character, it has probably come from the small intestine or the stomach.

The diagnosis of the causes of the bleeding has already been considered.

Prognosis.—The prognosis depends entirely upon the cause of the hæmorrhage. Great loss of blood may prove immediately fatal or may produce an extreme and very persistent anæmia. Sometimes in typhoid fever an intestinal hæmorrhage, if not too large, exerts a favorable influence upon the other symptoms.

Treatment.—Absolute rest in bed is imperative in cases of hæmorrhage of any considerable size. The diet should consist of cooled liquids. An ice-bladder should be placed upon the abdomen near the situation from which the blood is thought to have come. Injections of ice-water or of astringent solutions may be given when the blood comes from the large intestine. Ergot should be administered hypodermically if the symptoms are threatening. Opium is indicated to quiet peristaltic movements of the intestine, and turpentine or erigeron internally in large doses is sometimes useful to check hæmorrhage. In other cases, acetate of lead or gallic acid may do better. Stimulants and external heat, and hypodermic injections of ether and strychnine are indicated in collapse, and sometimes transfusion of blood or an intravenous injection of salt solution may be advisable.

AMYLOID DEGENERATION OF THE INTESTINE.

This alteration of the structure of the intestine is frequent in those who are the subjects of widespread waxy degeneration. It is common in phthisis and after any prolonged suppuration, particularly of the bones. It is also a very common development in syphilis, even without bone lesions.

It involves usually both the large and small intestine, but is especially well developed in the lower portion of the ileum and the upper portion of the colon. The mucous membrane is the coat principally affected, although, as a rule, the solitary glands and Peyer's patches are little, if at all, involved. The process begins in the smaller blood-vessels. In well-developed cases the mucous membrane is pale and thickened, and its appearance has been compared to that of wet wash-leather. The change, however, cannot be positively recognized macroscopically without the use of chemical tests. The application of iodine will give the characteristic mahogany-red reaction, and that of methyl-violet will produce a red-violet color in the amyloid material and a blue-violet tinge in the healthy tissue.

In very advanced stages the amyloid change may involve the whole thickness of the intestinal wall, and the mucous membrane may ulcerate.

The principal symptom of waxy disease of the intestine is chronic diarrhœa of moderate severity, generally unattended by pain. In the late stages of the disease hæmorrhage from the intestine may occur, and, though usually slight, may be very severe.

The diagnosis can only be made by observing that the causes of the disease are present or that amyloid changes are discoverable in other organs.

The fact that the intestine is evidently involved is a sign that the implication of the organs of the body is extensive, and that the disease is approaching a fatal issue. Treatment can only be palliative. The strength must be supported, the nourishment be concentrated and unirritating, and the diarrhœa or hæmorrhage controlled, if possible, by appropriate measures.

HÆMORRHAGIC INFARCTION OF THE INTESTINE.

This rather rare condition is due to *embolism of the superior mesenteric artery.* Blocking of the smaller branches probably has no serious result, since there is abundant collateral circulation. If, however, the main trunk or several neighboring branches of the artery are blocked, infarction follows before collateral circulation can be established through the inferior mesenteric and pancreatico-duodenal arteries.

The extent of intestine affected depends upon the size of the obstructed artery. If the embolus is in the main trunk, the greater part of the small intestine and the upper part of the large intestine are involved. The wall of the diseased portion, especially the mucous membrane, is swollen, œdematous, and infiltrated with blood. The mucous surface is covered with blood or bloody mucus. Necrosis of the mucous membrane may occur in places. Hæmorrhage may take place in the mesentery also. Circumscribed or general peritonitis is liable to develop.

The symptoms of infarction consist of severe colicky abdominal pain, free intestinal hæmorrhage for which no other cause can be found, and fall of temperature with other symptoms of collapse. Soon there develop tympanites and symptoms of peritonitis, and exceptionally the infarcted mesentery can be palpated through the abdominal walls. The blood from the bowel may be either bright-red, clotted, or tarry, and sometimes is not discharged at all. Besides these symptoms, there is liable to be present disease of the heart, which suggests an embolus and its origin, and emboli in other parts of the body are frequently found. The symptoms of acute hæmorrhagic pancreatitis are, however, so similar to those of infarction of the intestine that the diagnosis between the two affections is hardly possible.

The disease is a serious one, and almost always fatal. Death in collapse usually follows in a few days or a week. Instances of recovery have been reported, but there cannot but be some doubt about the correctness of the diagnosis in these cases.

The treatment is purely symptomatic. The effort must be made to sustain the strength by stimulants, to relieve pain with opium, and to check hæmorrhage with ergot and by enforcing absolute rest.

INTESTINAL INDIGESTION.

Just as there is a functional gastric dyspepsia, it seems probable that there may be an intestinal indigestion entirely independent of any structural lesion of the bowel itself. Such a condition might result from (*a*) the entrance into the bowel of chyme which has been imperfectly prepared by the stomach; (*b*) deficiency in the secretory or motor power of the intestine; (*c*) alteration in the biliary secretion; (*d*) deficiency in the secretion of pancreatic juice. The last two conditions are best described under the topics of Diseases of the Liver and of the Pancreas respectively.

As regards the first two causative conditions mentioned, there are no means of distinguishing, on the one hand, the cases in which the symptoms are produced by them alone without changes in the intestinal walls, and, on the other hand, the cases in which there is a mild catarrhal inflammation of the small intestine to which the symptoms of indigestion are secondary. It seems, in fact, probable that nearly all cases of indigestion are attended by more or less inflammatory catarrhal change.

ENTERALGIA.

Definition.—Intestinal pain of a neuralgic nature located in the intestine and dependent upon irritation of the sensory filaments of the intestinal nerves.

Distinctions between colic and enteralgia are often made which vary with the different writers. It is better to use them as synonymous terms until some definite points of difference can be determined upon, should such ever appear necessary.

SYNONYMS.—Enterodynia; Intestinal colic.

Etiology and Pathology.—As in gastralgia, to which the intestinal affection corresponds, the causes of the disease are dissimilar, and may be classified in different groups. There are certain predisposing causes, such as a neurotic temperament, hereditary tendencies, excessive mental work, business cares, improper hygiene of any kind, impairment of the general health, the existence of chronic debilitating diseases, and enfeeblement of digestion from any cause. Females are more predisposed to the affection than are males. It is not infrequent in children.

The active causes may be—1, local; 2, reflex; 3, dependent upon some disease of the central or peripheral nervous system.

1. The intestinal contents may abnormally irritate the terminal filaments of the intestinal nerves and produce pain. A large accumulation of hardened fæces acts mechanically in this way, for the intestinal wall with its nerves is greatly stretched and the terminal nerve-filaments are pressed upon and irritated. Meconium retained too long after birth produces what has been described as *colica meconialis*, and which is but enteralgia induced by a local cause.

Foreign bodies in the intestine, either those which have been swallowed or such as gall-stones, enteroliths, or masses of ascarides or tæniæ, produce enteralgia in a similar manner. Very often abnormal decomposition which has taken place in the contents of the stomach, or food which was unfit to eat, such as unripe fruit or sour milk, causes colic; sometimes by a chemical irritation of the nerve-endings, and sometimes by a mechanical irritation through pressure of the gas produced. This form of "wind colic" is an especially frequent one.

Sometimes individuals exhibit idiosyncrasies toward certain articles of diet, and suffer from pain whenever these are eaten. Nearly any article of food, though ordinarily healthful, may act in this manner in some cases, the kind of food which is harmful varying with the case. The colic is produced, however, with especial frequency by shellfish, fish, veal, acid drinks, ice-water, cold foods, and some fruits. Cathartics and various poisonous drugs may produce enteralgia by the direct local irritation of the nerves which they cause.

The various structural lesions of the intestine which have been described may produce enteralgia by the local irritation of the nerve-filaments. Many of the cases of intestinal obstruction exhibit severe enteralgia, the result of compression of the nerve-endings; and much of the pain of ulcers of any kind, of morbid growths, and of intestinal catarrhs is also of the nature of colic from local nerve irritation.

2. Enteralgia not infrequently arises reflexly from such causes as disease of the uterus, ovaries, liver, kidneys, spleen, and pancreas. It is also often seen in arthritis. For instance, the development of gout of the intestine

immediately after an attack of gouty arthritis is possibly produced in a reflex manner. Exposure to cold may be followed by intestinal pain arising reflexly from the chilling of the skin.

3. Enteralgia may result from such affections as organic diseases of the brain and spinal cord. Thus the crises of locomotor ataxia may involve the nervous system of the intestine. Hypochondriasis and hysteria are also central causes of enteralgia, and emotion may act in the same manner. Anæmia, malaria, lead, copper, and arsenic are also productive of enteralgia through their general systemic nervous action.

Symptomatology.—Pain in the intestine is the principal symptom of intestinal colic. It is usually seated in the neighborhood of the umbilicus, and may radiate thence over the whole abdomen or may shift from place to place. It is often associated with rumbling noises. The abdomen is sometimes retracted and hard, sometimes tympanitic. Occasionally coils of intestine are plainly visible in outline through the abdominal walls, and may be seen in active peristaltic movement. Often firm pressure diminishes the pain, and the patient frequently obtains relief by pressing the abdomen against the back of a chair or some other hard body or by lying upon the stomach. In some instances, however, the abdomen is excessively tender to pressure.

The attack comes on, as a rule, slowly, but may exhibit a very sudden onset. The severity of the pain varies from a dull ache to a sharp, most severe, cutting pain. It usually grows more and more intense, and may be so great that the face becomes pale and cold perspiration breaks out over the body. The pulse is usually hard and slow.

Various reflex symptoms often develop. Among them are hiccough, dyspnœa, palpitation, vomiting, tenesmus, strangury, retraction of the testicle or of the anus, priapism, seminal emission, cramps in various parts of the body, dizziness, syncope, and even general convulsions.

Diagnosis.—Although the diagnosis is often easy, it is often, too, attended by difficulties. There are some conditions which may very closely resemble enteralgia. Chief among these is rheumatism of the abdominal muscles. In this affection, however, the pain seems superficial, and even slight, and pressure and movement of the body increase it. It is more persistent, and does not exhibit exacerbations and remissions. There are usually, too, rheumatic manifestations elsewhere. Lumbo-abdominal neuralgia also resembles enteralgia, but the pain is unilateral and extends to the back, and the characteristic tender points can usually be detected. Nervous dermalgia occurs in hysterical subjects. It exhibits tenderness of the skin and evidences of hysteria. In localized peritonitis there is fever, and perhaps dulness on percussion and tenderness on pressure. In intestinal obstruction there is localized tenderness, the pain is more continuous, and there is obstinate constipation, while vomiting, often stercoraceous, is a prominent symptom. In uterine colic the pain is localized in the region of the uterus, and generally occurs at the time when the menses are expected. Hepatic colic exhibits pain chiefly in the right hypochondrium, and renal colic in the course of the ureter extending down to the pubes and thigh.

Prognosis and Course.—The attack lasts a variable time, sometimes only a few seconds, sometimes for hours or days. The prolonged cases usually exhibit less intense pain. Most frequently the attack consists of a series of exacerbations of acute pain, which follow each other more or less closely, and perhaps with continuous dull pain in the intervals. The disease may end gradually or rapidly.

The prognosis is almost always good, so far as temporary recovery is concerned. In very rare cases death has occurred. As regards the permanent cure and the avoidance of the relapses, which are so liable to occur, the prognosis depends entirely upon the ability to remove the cause.

Treatment.—The indications for treatment are,—first, to relieve the pain; and, second, to remove the cause. If the pain be very severe, its relief should be the first duty of the physician. Turpentine stupes or hot poultices to the abdomen are useful. Morphine by the mouth or hypodermically is indicated in the severe cases. Opium preparations may be used, even when constipation is present, since they relieve the spasm which is often the cause of the pain, and thus also allow the fæces to move onward. Carminatives find a useful place in the treatment of intestinal colic. Among them may be mentioned ginger, peppermint, capsicum, and oil of cloves. This class of remedies is especially indicated when the enteralgia depends upon the presence of gas. For the same reason chloroform, Hoffman's anodyne, and aromatic spirits of ammonia are of service.

The removal of the cause, when this is possible, is to be accomplished by appropriate treatment. For instance, a large accumulation of fæces must be treated by laxatives, and irritating food in the intestine is to be removed in the same way. Accumulations of gas in the bowel must be avoided by a suitable diet and the administration of remedies which will prevent fermentation. In cases exhibiting a persistent neuralgic tendency, the use of arsenic, of bismuth and pepsin, of dilute hydrocyanic acid with valerian, or of silver nitrate with small doses of opium and belladonna is to be advised.

DILATATION OF THE INTESTINE.

Dilatation of the intestine may occur as a symptom of any form of obstruction, as has already been stated, and intestinal indigestion, with the formation of large quantities of gas, is followed by the same result, although to a less degree. Chronic constipation may also be the cause of great dilatation, without any actual impaction of fæces developing. In some cases a condition of passive dilatation is itself the cause of the constipation.

The condition may be transient or chronic. The seat of the dilatation is usually the colon, although in rare instances the small intestine is the part affected. The whole of the colon may be distended, or, oftener, only the sigmoid flexure. Dilatation of this latter occurs especially when this part of the intestine is congenitally abnormally long.

Sometimes the distended bowel is so enormous that it fills the greater portion of the abdominal cavity, displacing the other viscera. Although the bowel in such cases is very commonly filled with fæces, yet this is not always the fact. In a case reported by Formad the man had an abdomen so huge and tympanitic, as a result of the enormous dilatation of the colon, that, profiting by his condition, he exhibited himself during life as a museum freak.

There is a rare affection, usually occurring in advanced life, in which dilatation of the rectal pouches takes place. These become permanently distended by hardened fæces and produce the symptoms of weight and uneasiness about the anus, with itching and pain after defecation.

The sacculi of the colon may also become distended by fæces in old persons, and enteroliths be formed in them.

CONSTIPATION.

Definition.—Prolonged retention of fæces, or expulsion of them which is habitually difficult or insufficient.

SYNONYMS.—Costiveness; Obstipation.

Etiology.—What constitutes a regular movement of the bowels cannot be absolutely stated. In the great majority of cases the bowels should be opened once a day and the stool be formed. In some persons, however, twice a day is the regular rule, and in others every other day or even less often is the normal habit, and cannot for them be called constipation.

There are a variety of causes of the affection. The condition may be a constitutional one—*i. e.* the patient may have been born with it. It is frequently hereditary. Women are more disposed to it than are men; this fact depending chiefly upon the constant neglect of a habit of regularity and upon the less active life led by them. Beside these causes, recurring pregnancies, the compression of the rectum by a turgid womb, the prolapse of a tender ovary, or the occurrence of the menopause tend to make women more the subjects of constipation than are men.

A sedentary life is a fruitful cause of the affection. All unfavorable hygienic influences of any kind which affect the general health favor its development. One very pronounced cause, as already stated, is the neglect of habits of regularity in defecation, as a result of which the susceptibility of the mucous membrane of the intestine is finally lost. The custom of taking purgative drugs in like manner gradually wears out the intestinal power. Certain diseases, as anæmia, chronic affections of the heart, lungs, liver, and stomach, wasting diseases, acute fevers, and weakness of the abdominal muscles from obesity or over-distention from pregnancy, are causes of constipation, most of them acting by enfeebling the muscular power of the intestine.

Many diseases of the intestine produce constipation, and in various ways. Painful affections of the rectum and anus render the patient unwilling to use the necessary effort to obtain a movement. Constipation may also be a result of the obstruction of the movement of the fæces in the intestine by tumors, stricture, impacted foreign bodies, and other obstructive diseases already described. Catarrhal conditions which might seem likely to be always attended with relaxation of the bowels may, on the other hand, favor constipation, probably owing to the excessive secretion of thick, sticky mucus which occurs in some cases. Atony of the bowel is one of the most frequent sources of constipation.

Gastric disorders frequently cause constipation by a reflex action. Loss of fluid from the body in any way is a not infrequent cause. Thus excessive perspiration in hot weather may produce constipation in those who at other seasons are of regular habit.

The nature of the diet used is a very prominent cause of the disease. Food that contains but little waste, or is insufficient in quantity, or which is coarse and has too much waste material, may be productive of constipation.

Pathology.—No lesions whatever may be found which are the result of constipation. In other cases, when the affection has been very persistent and extreme, there follow great dilatation of the colon, especially of the sigmoid flexure, and dilatation of the sacculi. This, as well as the production of stercoral ulcers and the lesions of impacted fæces, has already been treated of elsewhere.

Symptomatology.—The chief symptom is of course the undue retention of the fæces. Various secondary symptoms exist depending upon the constipation. The liability to these differs greatly with the individual. Many persons feel very wretched if a single day goes by without a passage from the bowels. Others, again, will go for a week or more without an evacuation and be in perfect comfort. In the average case debility, loss of appetite, headache, coated tongue, mental depression, insomnia, and a sense of fulness and discomfort in the rectum are frequent symptoms of constipation which has lasted from two to four days. The pressure of the hardened fæces in the rectum upon the uterus may produce uterine symptoms. Neuralgia of the sacral nerves may develop. The occurrence of nocturnal seminal emissions is a common result, and in children nocturnal enuresis may be produced in the same manner.

When a passage does occur, the large, hard masses of fæces distend the anus and leave it sore and irritated. Hæmorrhoids are a very common result of the necessary straining at stool.

If the retention of fæces be prolonged, the colon becomes full of masses which can be felt through the abdominal walls. The symptoms of intestinal obstruction may appear, while in other cases stercoral typhlitis develops, or, in still others, the hardened masses become channelled and a diarrhœa may be present, the results of the catarrhal condition set up by the constant pressure.

Prognosis and Course.—The chances for recovery from chronic constipation depend upon many circumstances—as, for example, the duration of the disease, the nature of its cause, and the state of the general health. There is little danger of any serious results developing, if proper care be taken; but many persons well advanced in life will always be of a constipated habit and will never be able to do without treatment. The prognosis of chronic constipation in infants is favorable. When a mixed diet is commenced, the constipation will very probably disappear.

Treatment.—In all cases of chronic constipation hygiene and diet should be the first consideration, rather than drugs. It is particularly important to cultivate a regular habit of evacuating the bowels. The patient should choose some hour of the day when there is plenty of time at his disposal, and retire and wait. No efforts at straining should be made. Very frequently after he has been sitting five or ten minutes at stool an evacuation will spontaneously take place.

Persons whose lives are sedentary must, in some manner, take a moderate amount of exercise. It is never difficult to secure this. Apparatus is not essential. Systematic, frequently repeated, deep diaphragmatic respirations, with contractions of the muscles of the chest, back, and abdominal walls, are valuable. Cold bathing and frictions act as a general tonic measure, and are often of benefit, as is the application of the faradic current to the abdomen. It is sometimes advisable to introduce one electrode into the rectum and apply the other to the abdominal walls. Massage of the abdomen is of the greatest benefit, the kneading being in the direction of the colon. A substitute for this is the rolling of a heavy metallic ball over the abdomen. The wearing of an abdominal bandage is of value in stout persons or in women whose abdomens are relaxed from childbearing. Diet is of the utmost importance. Food must be chosen which makes sufficient waste, without producing too much of it. Such vegetables as spinnach, lettuce, asparagus, tomatoes, salsify, celery, and the like are of value, and are frequently quite sufficient to ensure entire regularity of the bowels. Fruit, especially stewed, is of service. Stewed prunes are a very popular and effective dietetic remedy. Figs, stewed apples, raisins, and the like may be taken with advantage. There is a popular belief in the value of fresh fruit as a remedy for constipation, which often leads to increased digestive trouble from the excessive amount taken. Undoubtedly, however, in some cases, even a single raw apple or some other form of fresh fruit will obviate an otherwise troublesome diarrhœa. Oatmeal is an excellent food for the purpose in many cases. A glass of cold water, taken soon after rising, or on retiring, is sometimes quite effectual. In other instances a cigar smoked before breakfast attains the desired end.

Where drugs are required, strong purgatives are to be absolutely prohibited. Strychnine forms one of the most valuable remedies, since it gives tone to the enfeebled muscle of the colon. Belladonna may be combined with it, as may be ipecacuanha. Cascara sagrada is one of the most useful of laxatives, as it does not seem to lose its power or to take away the force of the intestinal muscle; consequently some sufferers with constipation depend for years upon small daily doses of elixir or fluid extract of cascara. Small doses of aloin or of podophyllin are also of value. An efficient preparation is a pill consisting of Ext. cascaræ sagrad., gr. 1; Aloin, gr. $\frac{1}{8}$; Ext. nucis vomicæ, gr. $\frac{1}{4}$; Ext. belladonnæ, gr. $\frac{1}{6}$, to be taken at night. Sometimes a pill of only one-half this strength is quite sufficient; and it is highly important to take only the smallest and mildest dose that will be effective.

In many cases the saline mineral waters taken in small dose before breakfast are of great value. Among the most useful and safe of these is Carlsbad sprudel salts, a teaspoonful of which may be taken in a half-pint of hot water before breakfast.

Many cases of constipation are associated with a lowered tone of system as well as with defective digestive secretions. A tonic regimen and tonic remedies which favor this are often useful. Olive oil or cod-liver oil may prove valuable. Pancro-bilin is at times useful.

In many cases, in addition to diet regimen, and the occasional use of a mild laxative, it proves necessary to resort to enemata to secure satisfactory results. A simple enema of cool or tepid water may suffice; or it may be strengthened by the addition of soap, salt, molasses, or vinegar. The cautious addition of nitric acid, from 10 to 20 drops in a pint, is very useful, especially in cases where there is mucous colitis, or where decided chronic catarrh of the lower bowel exists. It is important not to form the injection habit. The rectum readily falls into the way of not responding to the presence of an ordinary amount of fæces, but of requiring the stimulus of an enema. I have had patients who took one or more enemata daily for sixty years. Obviously they should be taken no more frequently than necessary, and the condition may begin to improve only when the effort to force a daily stool is abandoned. The character of the enemata may be changed. A small amount of glycerin in the form of a suppository, or of an injection (2 or 3 drachms with 1 or 2 ounces of water) may be efficacious. Where the rectum is very irritable and sensitive, and yet constipation is marked, I have known a half-pint of strong black coffee to be the most soothing and efficient injection. In cases where there is dilatation of the colon, with the tendency to form scybala, admirable results may be obtained from flushing the lower bowel two or three times a week with from 4 to 8 pints of warm water, which may contain a teaspoonful of salt or of borax to the pint.

NERVOUS DIARRHŒA.

In addition to the forms of diarrhœa already described as the result of various structural lesions of the intestine, the condition is not infrequently produced by purely nervous influences. Thus many persons have an attack of diarrhœa following anxiety, embarrassment, or emotion of any kind. In children it is not infrequently produced by fright. In a case on record, a surgeon suffered from an attack of diarrhœa before nearly every surgical operation which he was called upon to perform. It is seen not infrequently in hysterical, neurasthenic, and hypochondriacal cases.

Diarrhœa of nervous origin may also be seen in the course of certain nervous diseases. Thus, cases of Graves' disease often have attacks of it, and tabes may show itself in crises of this kind. Sometimes a very chronic form occurs in the latter disease. The menopause is not infrequently attended by diarrhœa of a nervous nature, as are diseases of the female genital apparatus. The peculiar idiosyncrasy shown by some persons in that certain articles of diet produce diarrhœa almost immediately after they are eaten is best explained on a nervous basis. All these cases are due to increased peristalsis or increased secretion, both of which are of nervous origin, and not at all dependent upon any catarrhal inflammatory process.

The treatment must be directed entirely to the nervous system.

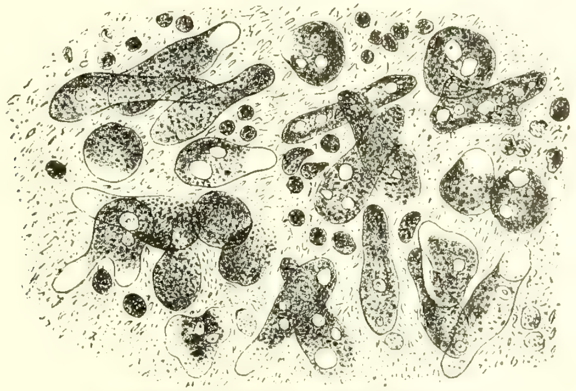
INTESTINAL PARASITES.

PROTOZOA.

A NUMBER of protozoan organisms have been found in the intestinal contents and intestines of man, the nature of some of which remains undetermined. There is doubt also as to the part which these organisms play in disease—whether they are in reality causes of disease, or whether the conditions favorable to their growth and reproduction are furnished by the intestinal affections with which they are associated.

AMŒBA COLI.—Lambl in 1859 described certain amœbiform organisms which he observed in the intestinal discharges of a child, but the amœba coli was first accurately described by Lösch in 1873. It is a round or irregular-shaped cell about .020 to .035 mm. in diameter (Fig. 66), consisting of a clear outer zone of protoplasm and granular substance within. Clear vacuoles are frequently observed, sometimes as many as seven or eight, and a nucleus and nucleolus may be visible. In the quiescent state the amœba is round, but ordinarily it is in active movement, projecting pseudopods on every side, often several at a time, but usually only one or two. When the surrounding conditions are unfavorable to its life, the organism becomes "encyst-

FIG. 66.

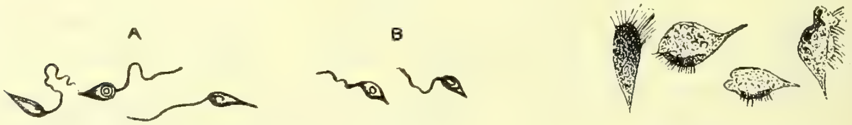


Amœba Coli in Intestinal Mucus (after Lösch).

ed." In this condition it is round or oval and quiescent, and is surrounded by a resistant outer covering. Cunningham, Grassi, and others have found amœbæ in the stools of healthy persons, but the amœba coli has been found most abundantly in dysentery, and is regarded by some writers as the cause of certain forms of this disease. For the destruction of these organisms Lösch used rectal injections of solutions of sulphate of quinine (1 : 5000). Other antiseptic remedies have also been employed.

Cercomonas intestinalis.—This is a pear-shaped organism, having at the narrow extremity a filament of about the length of the body, and a long flagellum, three times its length, at the broad end. A large and a smaller variety were described by Davaine, but those usually observed vary from .008 to .012 mm. in length. (See Fig. 67). They are actively motile, but may be held fast to epithelial cells or granular matter by the flagellum. The cercomonas is found in the glairy mucous stools of children, in typhoid fever, cholera, and various forms of diarrhœa. Lösch found it quite frequently in dysentery, and Cunningham in the excrement of animals or man when the reaction was alkaline. A similar, if not the same, organism was found in the collections about the teeth by Zunker. Injections of bichloride of mercury (1 : 30,000) have been used with good effect to destroy them.

Fig. 67.



Cercomonas Intestinalis. A, larger; B, smaller variety (after Davaine).

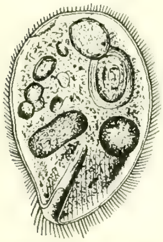
Trichomonas Intestinalis (after Zunker).

Trichomonas intestinalis.—This organism was discovered in the intestinal discharges of a case of typhoid fever by Marchand. It is somewhat larger than the cercomonas, being from .010 to .015 mm. in length and about .007 mm. in breadth. In repose it is almond-shaped, and is seen to have along one side of the body, more anteriorly than posteriorly, a membrane bearing vibratile cilia, like a comb. (See Fig. 67). The membrane and cilia are in constant motion. The posterior end of the organism is attenuated, but there is no flagellum. It contains irregular granules, and one or two vacuoles are present near the posterior end.

The trichomonas has been found in cases of typhoid fever, ordinary diarrhœa, and peritonitis. It is more frequently met with in acute cases than is cercomonas. Steinberg found a trichomonas in the mouth.

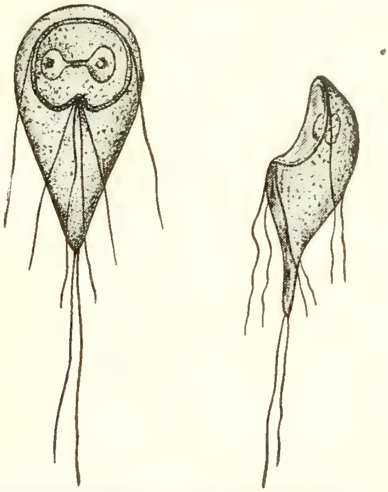
Balantidium coli or Paramœcium coli.—This organism (Fig. 68) has been found only in northern countries, especially at St. Petersburg, Dorpat, and Upsala. It is pear-shaped, from .07 to .10 mm. in length, and is surrounded by short cilia. The outer covering is clear, but the substance within is granular, and often encloses remnants of vegetable matter or blood-corpuscles which have been ingested. Two contractile vacuoles are frequently observed, and some observers have described a nucleus. At the anterior portion is a mouth or peristome; the anal aperture is scarcely perceptible. The natural host of the balantidium is the hog, and man is infected by drinking water contaminated by the encysted form of the organism. This is able to pass through the stomach without being destroyed. Balantidium is found only in cases of diarrhœa. Injections of tannic acid or vinegar are useful.

FIG. 68.



Paramœcium Coli (after Malmsten).

FIG. 69.



Megastoma Entericum from the Intestines of a Mouse (after Grassi and Schewiakoff).

Megastoma Entericum.—This organism was discovered by Lambl in 1859, but more thoroughly described by Gassi in 1879. It is irregularly pear-shaped, having at the broad anterior end a cup-like excavation situated obliquely. On the anterior lip of the excavation is found a pair of flagella, and on the posterior lip two pairs. The posterior end of the organism is somewhat drawn out or attenuated. (See Fig. 68). The size varies from .008 to .016 mm. The organism is found in the jejunum and duodenum, and in these positions is closely applied to the epithelial cells by its cup or excavation, like a cap. In the large intestine and in the stools smaller, oval, encysted forms are observed. Sometimes, when peristalsis is active, the ordinary active form may be rapidly carried downward, and may be found in the fæces. Lambl found the megastoma in enormous numbers in the mucus of infantile diarrhœa, and it has been noted by other observers in various conditions. Moritz found it in the stools of a perfectly healthy child.

PSOROSPERMIA.—Psorospermiæ are said to have been observed in one case by Slydlowski in the intestinal contents. Virchow, Eimer, and Klebs had previously described the post-mortem appearances of psorospermiasis in the human intestines. Psorosperms are recognized by their oval shape and the double contour of the outer coat. The protoplasm within may be uniformly filled with coarse granules, or it may be clear and contain a ball of fine granules in the centre.

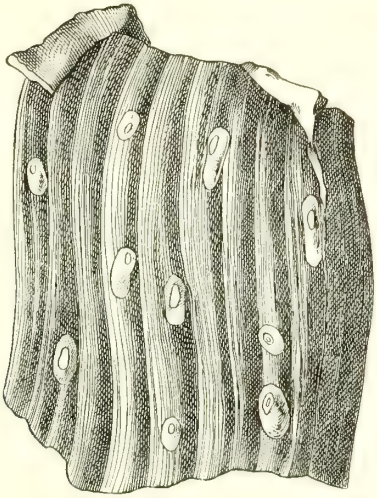
CESTODES.

Cestodes, or tape-worms, have been recognized since the time of Hippocrates, but their complete cycle of development remained obscure until recently. In their mature state, in which they constitute the familiar tape-worm, they occupy the intestinal canal of man or other vertebrates, while in the larval state they are found in the muscles, the liver, and other organs, but not in the intestines. For the full development it is necessary that the larval condition shall have been passed in an intermediate host.

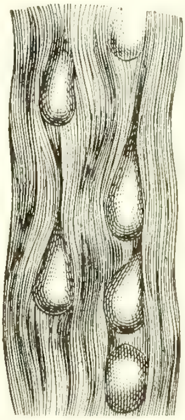
As ordinarily observed the tape-worm is a long, flat, soft, tape-like body, composed of numerous segments, the links or proglottides. At the anterior end there is a small head, provided with suckers or hooklets, by which it attaches itself to the mucous membrane of the intestines, and following this a slender neck, which gradually increases in breadth posteriorly, and ends in the fully-developed body. In reality, the tape-worm is not a single animal, but a colony of separate organisms, the segments each sexually complete or hermaphroditic. There is neither mouth nor anal orifice, but the animal is nourished by imbibition of nutritive fluid. The association of the separate segments is made somewhat more intimate by a pair of longitudinal vessels which commence at the head and extend through the body, one on each side. The segments near the neck are immature ; those farther down are matured by the development of a branched uterus and a complicated sexual apparatus which is self-impregnating and leads to distention of the uterus with eggs. The ultimate segments contain merely the uterus overfilled with eggs, the other sexual organs having undergone more or less complete atrophy. The segments are produced by constant growth and segmentation of the neck, and as they increase in size become sexually mature, and afterward completely ripe and distended with eggs. From time to time these ripe or mature segments are discharged, singly or several linked together, either before or after they have discharged the eggs, with the intestinal contents.

The eggs or the segments after discharge may remain alive for several days in the excrement or in water into which it has been deposited, but they finally find their way into the intestines of some animal, the intermediate host, feeding where the infected material has been deposited. In the stomach of the animal in question the outer covering of the egg is digested, and the embryo or proscolex is set free. By means of six chitinous spicules with which it is armed the embryo is enabled to perforate the intestinal walls, and by direct migration or through the blood it makes its way to the muscles, to the lungs, liver, or other organs. At the final destination the hooklets are lost, and the larval form or scolex is developed. This consists of a head like that of the adult tape-worm, with a short neck, inverted into a cyst-like sac, and the whole surrounded by a connective-tissue capsule resulting from reactive inflammation in the surrounding tissues. Flesh containing the scolices of tape-worm is said to be measled, and the sacs themselves are known as measles. These are small oval or elliptical cyst-like bodies (Fig. 69), which not rarely present a small depression at the point at which the head or scolix is situated. The embryonal animal in the measles undergoes no further development, but after a period of months or years may degenerate, becoming atrophic or calcified. If the measled flesh of animals is eaten in an uncooked or insufficiently cooked condition, the scolex becomes liberated, attaches itself to the mucous membrane of the intestine, and becomes developed into the mature tape-worm. As a rule, but one tape-worm is developed, but exceptionally several are found, and in one case Kleefeld found forty-one. Laker described a case in which fifty-nine heads were found in a mass enclosed in the coils of a fully-developed worm; and it seems not improbable that partial development of this nature may occur more frequently than is at present believed to be the case. Two species may occasionally be observed at the same time, and tape-worms are often associated with other forms of intestinal parasites, such as ascaris, oxyuris, trichocephalus, and anchylostomum.

Fig. 70.



Cysticercus Cellulosæ, natural size (after Stein).



Cysticercus Tæniæ Saginatæ, magnified two diameters (after Stein).

Though, as a rule, man is the host of the tape-worm in its adult or mature form, the larval condition is also at times observed. In the case of the Tænia echinococcus man is affected only in this way, the mature worm occurring in the dog. Occasionally, however, cysticerci of the Tænia solium are found in man himself, and sometimes give rise to serious conditions.

There are three important varieties of tape-worms—the Tænia saginata, the Tænia solium and the Bothriocephalus latus, which will require full description, and a number of others which have been met with occasionally and which therefore require only brief notice.

Tænia saginata.—Synonyms.—Tænia mediocanellata; Unarmed tape-worm; Beef tape-worm.

This is by far the commonest form of tape-worm, and is derived by man from the ox, in which the larval condition, cysticercus Tæniæ saginatæ, is generally found. It varies in length from six to twenty-four feet, and as many as twelve hundred segments have been counted in a single worm. The head is of about the size of a yellow mustard-seed, and is somewhat quadrate and flattened on the top. It is provided with four suckers, by which the animal retains its position in the intestine. (Plate V, Fig. 1). Following the head is the slender, often somewhat flattened, neck, which merges into the body. The segments anteriorly are broader than long, but the fully mature are often twice as long as broad. (Plate IV, Fig. 1). At one margin of each segment and irregularly alternating may be seen a slight prominence or papilla, in which is the external genital aperture. The uterus consists of a median tube giving off fifteen or twenty short, transverse, and branching tubes. (Plate VI, Fig. 1). The eggs may be extruded from the uterus in the intestine, or after the segment has been discharged, or by its own motile power has escaped from the bowel. The eggs are exceedingly numerous, are of a brown color, of oval shape (Plate V, Fig. 1), and vary in length from .035 to .039 mm. In the ox the embryos contained within the egg are developed into cysticerci, which may be found in the muscles and in the liver or lungs. The relation of the beef-measles to tænia saginata was demonstrated by Perroncito, under whose direction students ate of the uncooked meat. Cysticercus Tæniæ saginatæ has also been found in the giraffe.

Tænia solium.—Synonyms.—Armed tape-worm ; Pork tape-worm.

This form was formerly regarded as much more frequent than is really the case, from the fact that Tænia saginata was confounded with it. It varies in length from five to ten feet, and is composed of about eight hundred segments. The head is spheroidal, about the size of a pinhead, and usually darkly pigmented. Anteriorly it bears a papilla or rostellum which is armed by a double row of hooklets, each row containing fifteen or sixteen. (Plate V, Fig. 2). At the sides of the head are four suckers, as in the beef tape-worm. The neck is short and thread-like, ending in the immature segment of the anterior part of the body. The proglottides themselves are broader than long anteriorly, but posteriorly are quadrate, or even a little longer than broad. (Plate IV, Fig. 3). The uterus consists of a median tube with six to twelve lateral branches, which are not so minutely subdivided as in the case of the beef tape-worm. (Plate VI, Fig. 2). The segments are not so frequently discharged as are those of Tænia saginata, and generally there are several together. Sometimes, however, solitary segments are expelled with the fæces or escape from the anus by their own motile power. The eggs of Tænia solium resemble those of the beef tape-worm, but are more spherical and somewhat smaller, being from .030 to .035 mm. in length. (Plate V, Fig. 2).

The pork tape-worm, as its name implies, is derived from the hog, and the larval condition is known as the Cysticercus cellulosæ. Measled pork is more common than measled beef, but as the latter is more frequently eaten raw or only partially cooked, the beef-worm is the common species in man.

PLATE IV.

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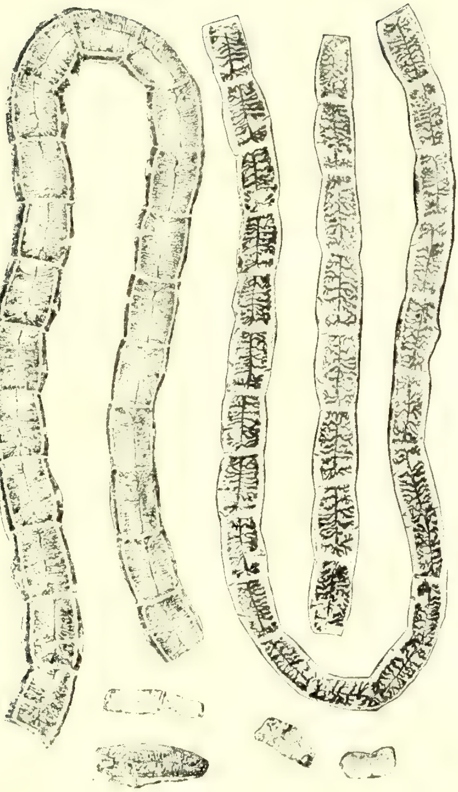

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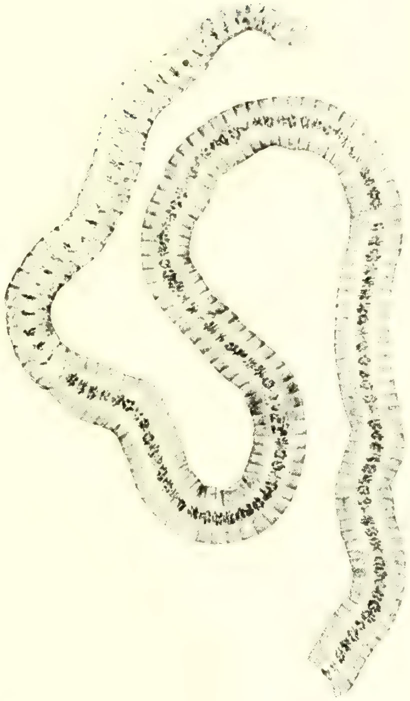

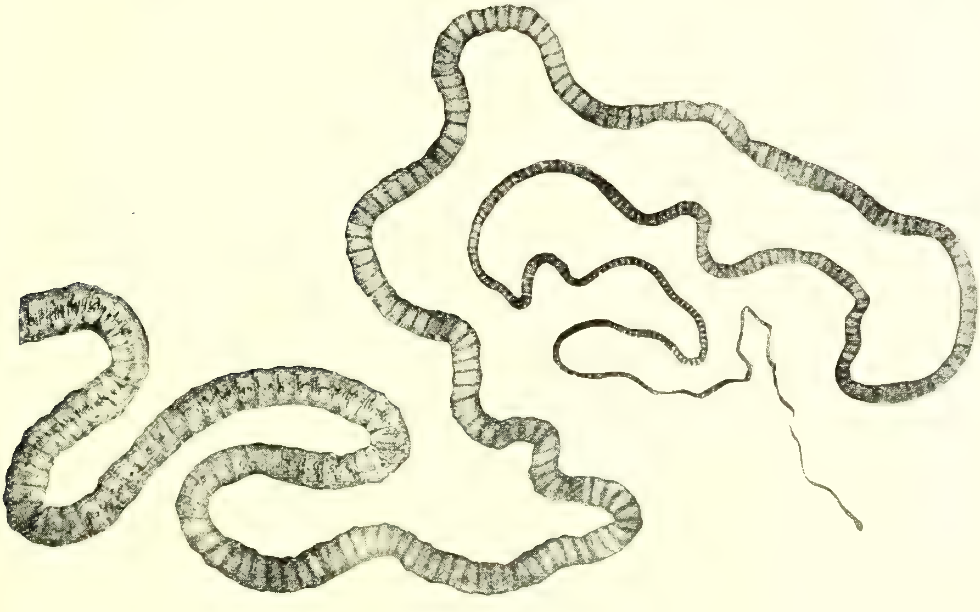

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Fig. 1.—Segments of Tænia Saginata.
Fig. 2.—Head and Segments of Bothriocephalus Latus.
Fig. 3.—Head, Neck, and Segments of Tænia Solium.
(*After Stein.*)

Sometimes the larval condition is found in man, resulting from the ingestion of eggs or segments, or possibly from introduction of segments into the stomach from the intestines through vomiting. The cysticerci are generally found in the subcutaneous tissues or in the muscles, but may be in the brain or cord or in the eye, in which case serious symptoms are produced. Though the hog is the intermediate host from which man, as a rule, derives the armed tape-worm, the measles of this species have also been found in the dog, deer, bear, monkey, and, according to some authors, in the sheep.

Certain other species of Tænia occasionally infest the intestines of man, though of rare occurrence. Some of them are armed with hooklets like the pork-worm, others unarmed like the beef-worm.

Those resembling the Tænia solium are the following:

TÆNIA NANA, the dwarf tape-worm, was discovered by Bilharz in 1851, but has recently been observed a number of times in Europe. It is about half an inch in length, composed of about one hundred and fifty to one hundred and seventy segments, and is armed with twenty-two or twenty-four hooklets in a single row upon the rostellum. It is usually present in considerable numbers.

TÆNIA ELLIPTICA.—This form and Tænia cucumerina are probably identical. It is a frequent parasite of the cat and dog, but is very rare in man. It is about four inches or a foot in length, composed of elliptical segments, and has a prominent rostellum armed with sixty hooklets.

TÆNIA FLAVO-PUNCTATA is a small species, eight to ten inches long, and distinguished by a yellow spot upon the sides of the proglottides. The head has never been observed. It was described by Weinland in 1858, and in 1884 Leidy made the second observation.

TÆNIA MADAGASCARIENSIS.—This species is imperfectly known. But a few fragments have been found and described.

Forms resembling Tænia saginata:

TÆNIA TENELAL is a tape-worm about three feet in length. It is probably derived from the sheep.

TÆNIA ALGERIANA was found by Redon in Algiers. It is derived from the sheep.

TÆNIA OF THE CAPE OF GOOD HOPE.—This form is imperfectly known, and was thought by Davaine to be an anomalous Tænia saginata.

TÆNIA NEGRE.—Davaine described under this name a tape-worm of dark brownish color. No other observation has been made.

The next genus of tape-worms, Bothriocephalus, was formerly confounded with Tænia, though the distinctions are now well recognized and clearly drawn.

BOTHRIOCEPHALUS LATUS. — SYNONYMS. — Dibothrium latum; Broad tape-worm.

This is the largest species of tape-worms observed in man. The length varies from fifteen to twenty-seven feet, and as many as 4133 segments have been counted in one worm. The breadth is frequently three-fourths of an inch. The head is almond-shaped, and is provided with two elongated, elliptical grooves or suckers (bothridies). (Plate V, Fig. 3). There are no hooklets. The neck is short, and merges into the segments of the body. The width of the proglottides is two to four times their length. (Plate IV, Fig. 2). The uterus consists of a group of radiating pouches or tubes presenting a rosette-shape, and the genital pore, unlike that of the Tæniæ, opens at the centre of the flat surface of the segment. (Plate VI, Fig. 3). The segments are not discharged separately, but together in portions containing a number of links.

The eggs are of oval shape and brownish color, and are from .06 to .07 mm. in length. The shell at one pole contains a lid by which the embryo is enabled to escape. (Plate V, Fig. 3). Like the tænia the embryo is armed with six chitinous spicules, but these are subsequently lost. The intermediate host of Bothriocephalus is not so certainly known as those of the beef and pork tape-worm, but the measles are probably those which have been found in the pike, trout, and eel-pout, and by feeding which Braun produced tape-worms in dogs and cats scarcely distinguishable from the Bothriocephalus of man. Bothriocephalus is common in Switzerland, Holland, Belgium, and especially in the Baltic provinces, where these fish are consumed. I have seen but one or two instances in Philadelphia, and these in the persons of immigrants.

BOTHRIOCEPHALUS CORDATUS is a rare form of tape-worm, attaining a maximum length of three or four feet. It was described by Leuckart as a common form infesting the dog in Greenland, and has been observed in man. The head is short and large, flattened laterally, and bears on each side a deep longitudinal groove or sucker.

BOTHRIOCEPHALUS CRISTATUS was twice observed by Davaine. It is from six to nine feet in length. The head is pointed anteriorly, and presents two elevated, longitudinal crests situated laterally.

Etiology.—Man becomes the host of the mature tape-worm in all cases by eating the uncooked or insufficiently cooked flesh of animals containing the proscolices. Habits of eating, therefore, play an important part in the etiology. The fact that uncooked beef is more frequently eaten than raw pork has already been cited as the explanation of the prevalence of Tænia saginata.

Children and women are more frequently affected than male adults. Certain occupations likewise increase the liability from the habit of eating raw meat. This is seen in the frequency of tape-worm among butchers and cooks. In St. Petersburg the use of fresh meat in the treatment of infantile diarrhœa is said to have increased the frequency of tape-worm; and, according to Bérenger-Féraud, it has increased in Western Europe, owing to the importation of beef and cattle from the Mediterranean basin.

No part of the world is exempt, but in certain countries, as in Abyssinia, where raw meat is extensively used, tape-worms are extremely common. The Bothriocephalus is found only in certain restricted localities near the Baltic Sea and in other portions of Europe.

Seat and Morbid Anatomy.—The head of the tape-worm is situated at or near the duodenum, and the body lies in loose coils in the upper part of the

PLATE V.

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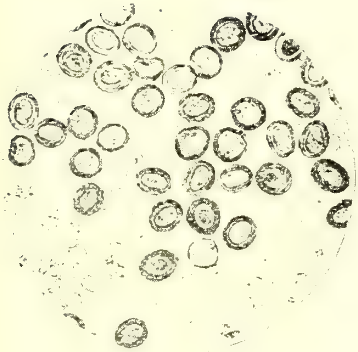

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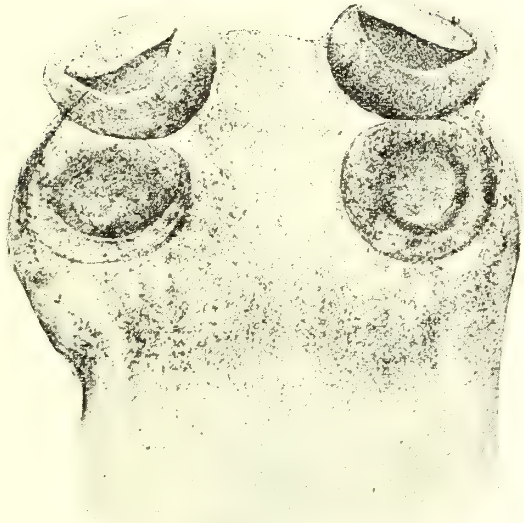

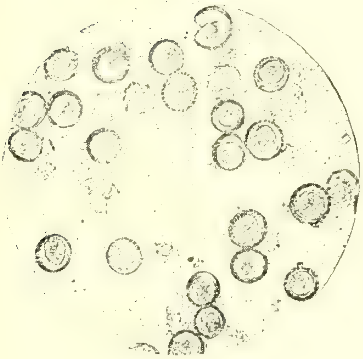

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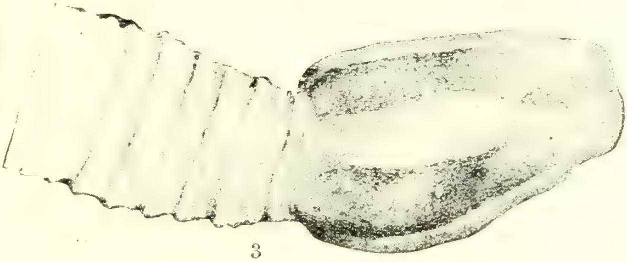

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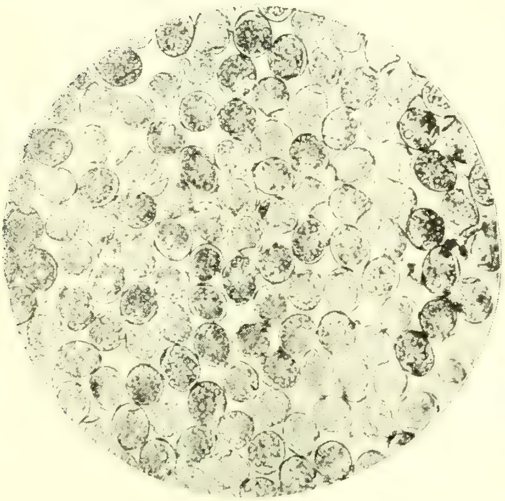

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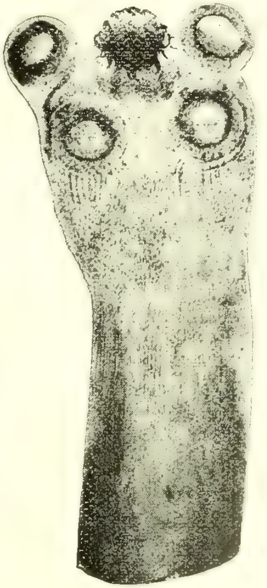

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FIG. 1.—Head and Mature Eggs of the Tænia Saginata. Eggs magnified 300 diameters.
FIG. 2.—Head and Mature Eggs of the Tænia Solium. Eggs magnified 300 diameters.
FIG. 3.—Head and Mature Eggs of the Bothriocephalus Latus. Eggs magnified 150 diameters.

(*After Stein.*)

intestines. Sometimes the posterior part of the worm is stretched out at length in the intestines; at other times the whole animal is closely coiled up, and may give rise to serious obstruction of the intestines. Loose portions of worm or separate segments are frequently found in the intestinal canal down to the rectum. Occasionally portions of the worm are found in the stomach, but this is never the normal situation, and presages its early destruction. In this way, however, man may become infected with the larval form from an adult worm within his own intestines. The mature tape-worm sometimes remains alive for years, and in one instance was known to have existed for thirty-five years.

Sometimes destructive lesions are found in the intestinal walls, such as ulceration, or even perforation, and these have been ascribed to the action of the parasites. Other authors, however, regard them as accidental and in no wise due to the tape-worm. Cases have been reported in which abscesses or fistulous communications have furnished a point of egress for the worm by way of the bladder and urethra, or even through the abdominal walls. In a number of instances portions of the worm have been found in the peritoneal cavity. More frequently parts have been discharged from the mouth in vomiting. Spontaneous discharge by the rectum may occur during febrile diseases like typhoid fever or phthisis, in dysentery, or after excessive indulgence in alcohol. Perhaps in some cases disease of the worm itself occasions its extrusion. A common abnormality or disease is that known as fenestration, in which each segment presents a perforation at the centre of the broad surface.

Symptomatology.—In many cases individuals infested with a tape-worm present no symptoms at all until the segments are discovered and attention is called to the existence of the condition. The links may be discharged with the stools, or, in the case of the beef-worm, not infrequently escape from the anus spontaneously. In the case of Bothriocephalus separate links are rarely found, but from time to time a chain of several segments is discharged. The links are sometimes expelled through the stomach and mouth in severe paroxysms of vomiting, and in case of the pork-worm this carries with it the danger of infection with cysticercus.

Local abdominal symptoms are, however, frequently present, and may be prominent. There may be merely an uneasy sensation about the umbilicus, or in other cases severe attacks of colic. The pain in the latter may be high up in the region of the stomach or low down in the pelvis; as a rule, however, it is situated about the navel. Pain is, as a rule, more urgent before meals, and may disappear after eating.

The appetite is sometimes inordinate, and it is striking that the patient grows progressively more anæmic and emaciated though the appetite remains good. I have observed extreme bulimia in several instances. In other cases there is complete anorexia. Nausea and vomiting may still further interfere with proper nutrition, and salivation of a distressing nature is occasionally observed. The bowels may be constipated or relaxed, and sometimes these conditions alternate. Some patients present the symptoms of gastro-intestinal catarrh, and these may continue for years, the real cause remaining undiscovered. If the head of the tape-worm be attached in the fossa of Vater, it may, as in a case reported by Moreau, cause jaundice and painful enlargement of the liver. In a case recorded by Letulle there was ascites and an icteroid hue of the skin.

Besides these local symptoms there are numerous manifestations regarded as of reflex origin, but doubtless in some cases these are due to the psychic effect resulting from the knowledge of the existence of the tape-worm.

Vertigo, spasms, and pruritus, especially constant itching of the nasal mucous membrane, are frequent symptoms. Headaches, hiccough, syncopal attacks, and inequality of the pupils have also been observed. Not uncommonly more serious nervous symptoms are observed, such as delirium, transient palsies, chorea, and convulsions. The latter are especially common in young children, and may be distinguishable only with much care from idiopathic epilepsy. As a rule, however, the onset of the fit is less sudden, severe falls and injury less common, and the duration is longer. Complete recovery may follow discharge of the worm. Hypochondriasis, and even verminous mania, have been described. Reflex disturbances of the respiratory organs may be manifested by cough, dyspnœa, or asthmatic attacks. Temporary aphonia is a rare symptom.

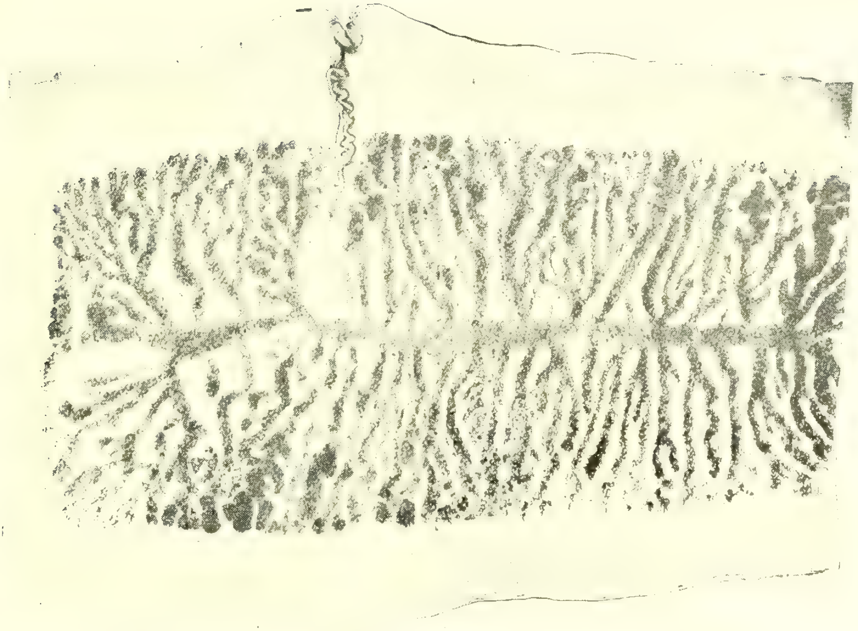
In cases of Bothriocephalus latus the symptoms above described may be present, and may continue until the worm is discharged. In some, however, after a variable length of time the patient suffers with diarrhœa, nausea, vertigo, palpitation of the heart, and becomes progressively more and more anæmic. The palpitations increase, œdema of the feet or of the whole body may supervene, and there may be fever, increasing diarrhœa, syncopal attacks, and profound prostration. The picture presented in these cases is indistinguishable from that which is seen in progressive pernicious anæmia. A few cases of this nature have resulted from the presence of the Tænia saginata.

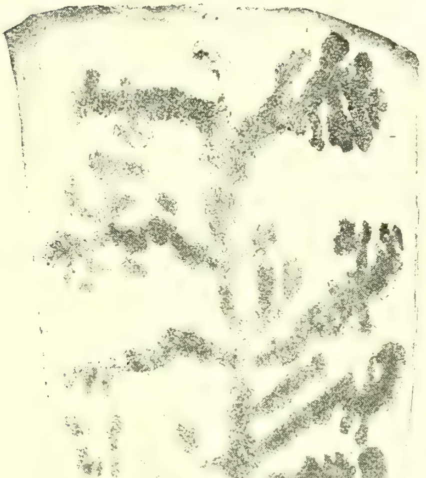
Symptoms due to cysticerci of Tænia solium are sometimes most urgent, depending upon the part of the body invaded. Frequently they are found in the subcutaneous tissues and muscles, and cause great muscular stiffness and general pain, with painful subcutaneous nodules. Sometimes the measles invade the eye, and may occasion opacity of the transparent media or lens, detachment of the retina, and irido-choroiditis, and generally more or less complete loss of vision. Cysticerci in the brain or cord may occasion epileptiform convulsions, spasms of various muscles, palsies, mental troubles, and various other symptoms depending upon the locality of the growth.

Diagnosis.—As a rule, the diagnosis of tape-worm is not possible until the ripe segments are discharged or escape from the anus spontaneously. Care must be taken, however, by the physician that he himself inspect the suspected links, for vegetable tissues and shreds of membrane in cases of mucous colitis are frequently mistaken for tape-worm. If portions of the worm have ceased to be discharged, mild purges will sometimes cause a reappearance and aid the diagnosis.

PLATE VI.

1





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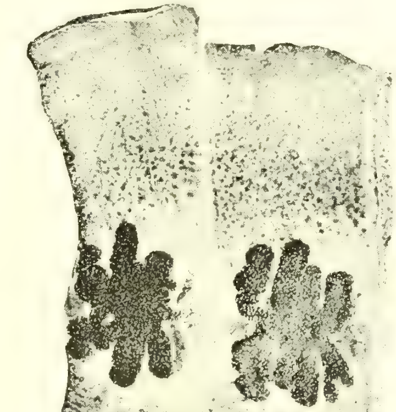

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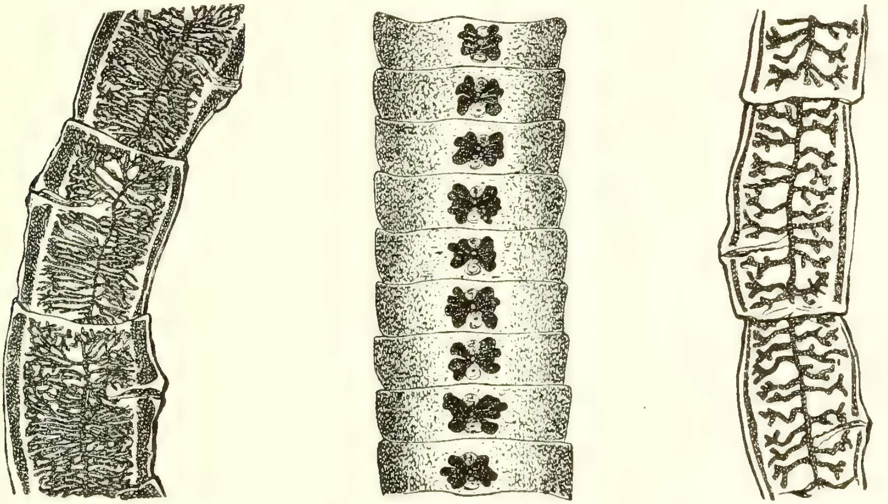
Figs. 1 and 2.—Segments showing Uterus of Tænia Saginata and Tænia Solium respectively. Magnified 8 diameters.

Fig. 3.—Segment showing Uterine Rosette of Bothriocephalus Latus. Magnified 10 diameters.

(*After Stein.*)

If segments are obtained, the diagnosis of tape-worm is certain, and in addition the particular variety may be determined. Bothriocephalus is distinguished by the fact that a number of links are usually discharged together, and that the genital pore is found on the broad surface and not at the edge of the link. If the segment be partly dried and pressed between two plates of glass, the rosette-like arrangement of the uterus is detected. Tænia saginata is distinguished from Tænia solium by the greater length of the links compared with the width, and by the greater number of branches of the uterus in the former than in the latter.

Fig. 71.



(1) Segments of Tænia Saginata (after Stein). (2) Segments of Bothriocephalus Latus (after Stein). (3) Segments of Tænia Solium (after Stein).

In some cases the diagnosis has been made by finding the eggs on microscopic examination of the stools. This method is especially valuable in cases of Bothriocephalus, in which the links are discharged at somewhat longer intervals than in cases of tæniæ. The egg of the former species is distinguished by its greater size and by the lid or operculum. Those of Tænia solium are smaller than those of Tænia saginata and more spherical, but the diagnosis in this case is extremely difficult.

Prognosis.—The prognosis in cases of tape-worm is always good, though the worm is often exceedingly difficult to remove. In those instances of Bothriocephalus in which advancing anæmia and exhaustion mark the case, the outlook is bad unless the parasite is promptly removed. Cysticerci of Tænia solium may be wholly latent and harmless when few in number and situated in the subcutaneous tissues or muscles, but become dangerous parasites in the eye or nervous structures.

Treatment.—The prophylaxis of tape-worm requires both individual efforts and regulations on the part of the State. Only food which has been properly cooked should be used, and habits of cleanliness of all kinds on the part of those whose occupation requires the handling of raw meat must be promoted. The official inspection of all animals, and of the meat after it has been prepared for sale, has in some countries produced a noticeable decrease in the prevalence of the disease.

Remedies which are used for the expulsion of tape-worm are administered without regard to the particular species. The tænifuges for the most part benumb the parasite temporarily, and must therefore be followed by a purge to expel it from the bowel.

Preparatory regulation of the diet is necessary, so that the intestines may be as empty of food as possible. The general condition of the patient will have weight in determining how rigid the restriction of diet may be. In cases of weak persons, such as those having phthisis, the treatment, if admissible at all, must be conducted with the greatest care, lest the patient's strength be exhausted. In vigorous subjects two or three days' restriction of the diet to milk or gruels, with mild purgation, is advisable.

The most generally useful remedy is the oleo-resin of male fern, which is in reality an ethereal extract of the drug. A half drachm or drachm of the remedy is given in the morning after two days' restriction of diet, and in the evening a brisk cathartic, such as castor oil, should be administered. Sometimes calomel is given in combination with the oleo-resin. The patient should remain abed after the administration of the remedy, to avoid syncope and other unpleasant effects of large doses of the drug. If the worm is partially expelled, no traction should be made upon the external portion, lest the body be torn off and the head be allowed to remain. The treatment may be considered successful if the whole worm is obtained, but if the head be retained in the intestines, it will rapidly grow again to its full size.

The decoction of pomegranate-rind has been used, and is an efficient though unpalatable remedy. Tanret, however, has obtained two alkaloids, pelletierine and isopelletierine, which appear to be the active principles of the drug. The method of using these drugs, as employed by Dujardin-Beaumetz, is as follows: In the evening a mild purge is administered, and at supper only a cup of milk is permitted. The next morning 30 centigrammes of sulphate of pelletierine or isopelletierine are administered in a solution containing 50 centigrammes of tannic acid, and followed by a glass of water in ten minutes and a brisk purgative in half an hour. This remedy is sometimes found efficient when all others have failed, but has the disadvantage of sometimes causing toxic symptoms—pallor, vertigo, disturbances of vision, cramps, vomiting, and great prostration. The cost of the remedy is also a disadvantage.

Pumpkin-seeds are valuable, especially in children, and are perfectly harmless. An ounce or two beaten up with sugar and water form a not unpleasant electuary, which is given in the morning and followed after a few hours by a purgative.

The infusion of koosso is a powerful remedy, but is apt to provoke nausea and vomiting. Koossin, in doses of twenty or forty grains, may be administered in a wafer instead of the infusion, and is more readily taken.

Other remedies have been used, but are less certain and more unpleasant than those named.

NEMATODES.

SYNONYMS.—Thread-worms; Round-worms.

Among the nematodes are included some of the most important and widespread of all the intestinal parasites of man. The forms which are most commonly met with are Ascaris lumbricoides, the common round-worm; Oxyuris vermicularis, the seat-worm; Anchylostomum duodenale; and Trichina spiralis. The last named gives rise to a group of general symptoms which has been designated as trichinosis, and which has been considered among the general diseases in Vol. I. Besides these forms there are rarer and less important round-worms, which require only brief description. A few species of trematodes, or fluke-worms, are also met with in the intestinal canal, but are infrequent parasites of man, and are therefore considered with the rarer forms of round-worms.

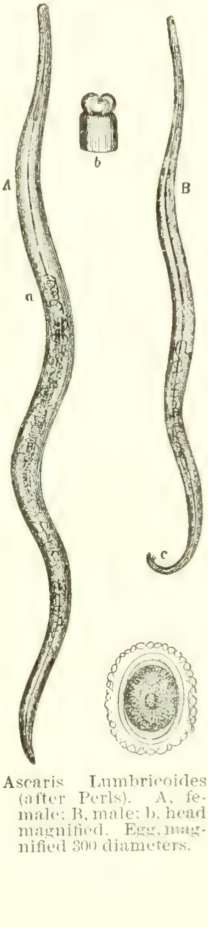
ASCARIS LUMBRICOIDES.—The common round-worm, Ascaris lumbricoides, is a frequent intestinal parasite in all parts of the world, excepting some of the colder countries of the extreme north, where it is rarely seen.

It is easily recognized by its cylindrical shape, which gives it an appearance not unlike that of common earthworms. The body is rounded, but marked by four longitudinal striæ and by innumerable parallel transverse striations. Both ends, but especially the anterior, are attenuated and more or less pointed. The female is from twelve to sixteen inches in length, and the male about eight inches. The body is of a grayish or pinkish color. The head is somewhat spherical, and is provided with three papillæ or elevations surrounding the mouth. The caudal end of the male is sharply curved toward the ventral surface, forming a hook-like extremity, in which is situated the cloaca, guarded by two chitinous hooks, the organs of fixation. In the female the caudal end is straight, and presents on the ventral surface the anal orifice. The genital opening is on the ventral aspect at the junction of the anterior and middle third.

The eggs, which are produced in immense numbers, are easily recognized. They are from .05 to .06 mm. in diameter, and are filled with granular contents. The shell of the egg is dark-colored and resistant, and is enveloped in an outer covering of albuminous material, which presents numerous prominences, giving rise to a moruliform appearance.

The cycle of development of the round-worm is still

FIG. 72.



Ascaris Lumbricoides (after Perls). A, female; B, male; b, head magnified. Egg, magnified 300 diameters.

imperfectly known. Attempts to develop it by feeding the eggs have always failed, so that it was held that an intermediate host is necessary, or that the embryo must be first developed outside the digestive canal of man. Linstow believed the intermediate host to be the common myriopod of gardens, Iulus guttulatus, but his view is probably incorrect. The opinion of most authorities at present is that the worm is developed directly from the embryo, which has been taken into the stomach in impure water or otherwise.

Etiology.—Ascaris lumbricoides is exceedingly common in warm climates, especially in the East, where habits of cleanliness are less rigidly observed than in other countries. In portions of Asia and Africa almost every person is infested at one time or another. Sometimes it prevails in almost epidemic fashion, as was the case at Göttingen in 1760.

Women and children are more frequently affected than men. The frequency among the insane in asylums is often noted.

Cleanliness and the avoidance of impure water or food are the manifest prophylactic measures which limit diffusion of the parasite.

Seat and Morbid Anatomy.—The natural seat of the round-worm is in the small intestines. There may be but one or a few, and in these cases no pathological changes may be present. In some cases, however, immense numbers of worms are found, even as many as five thousand, and in these more or less irritation and inflammation of the intestinal mucous membrane are apt to be induced. Occasionally cases are observed in which great numbers are massed together and give rise to intestinal obstruction.

From time to time one or more are passed with the stools, or they may migrate from the intestines and make their appearance through other channels. Not infrequently they are discharged from the mouth in the act of vomiting. Less frequently they may creep through the œsophagus to the pharynx and nasal cavities, and escape through the anterior nares. In rare cases they have been known to enter the larynx or bronchi, and to lead to suffocation or to pneumonia and gangrene of the lungs; or they may penetrate the Eustachian tube or the lachrymal duct.

Serious disturbances sometimes result from migration of the lumbricoids into the bile-ducts. In these cases more or less complete obstruction may occur, and cholangitis, or even abscess of the liver, may result. The intestines may be perforated, and the worms may escape into the peritoneal cavity, or externally through the urinary and genital passages, or by a fistulous opening through the abdominal walls. Sometimes round-worms are found in the pleural sacs, and even in the cavity of the pericardium.

Lumbricoids may occur alone, but are often associated with seat-worms, with whip-worms, or with tape-worms.

Symptomatology.—As in the case of tape-worms, there may be no symptoms at all, and the existence of worms is unsuspected until one is accidentally discovered in the stools or more rarely in matters vomited.

Gastro-intestinal symptoms are usually prominent. Abdominal pains of colic-like nature or other abnormal sensations are common. The bowels are irregular, sometimes constipated, at other times relaxed, and there may be attacks of violent diarrhœa. Mucous colitis, intestinal ulceration, and even perforation, are met with occasionally, and give rise to urgent symptoms.

The patient's breath is fetid; the appetite is frequently ravenous, and there may be a desire for unwholesome foods or various unpleasant substances, such as clay or chalk. In other cases the appetite is entirely lost. The patient grows anæmic and thin, and dark rings are frequently seen around the eyes.

Reflex disturbances, like those which occur in case of tape-worms, are often noted. Itching about the nares leads to picking of the nose, and there may be general pruritus. The sleep is frequently broken, and the child tosses restlessly or grinds the teeth. Severe nervous symptoms, such as vertiginous attacks, epileptiform convulsions, and chorea, sometimes occur, but are less frequent than has been supposed. The general symptoms are believed by Huber to be due to a poisonous substance elaborated by the lumbricoid.

Severe disturbance of the stomach may occur when the parasites migrate from the intestines through the pylorus. In this case vomiting and discharge of lumbricoids from the mouth is not uncommon; but vomiting may be induced in a reflex manner and the worms may be forced into the stomach by the violent contractions. In a case reported by Fauconneau-Dufresne one hundred and twenty-five round-worms were vomited by a boy in a single day. Intestinal obstruction due to round-worms sometimes occurs, but is extremely rare.

Jaundice, enlargement of the liver, and fever may result from obstruction of the biliary duct and cholangitis. If the lumbricoid enters the larynx through the pharynx, violent spasm and suffocation may result, or the worm may migrate into the bronchial tubes and occasion pneumonia or gangrene of the lungs.

Diagnosis.—Some cases are entirely latent and could only be discovered by microscopic examination of the stools. The eggs are so numerous and so easily recognized that the diagnosis can be made in this manner, even though the parasites themselves have never been discharged. As a rule, however, the diagnosis is not made until a worm is passed in the stools or is vomited. In children restlessness, picking at the nose, and indefinite disturbances of the stomach and bowels should always lead to careful examination of the stools. A mild purgative may be given to favor discharge.

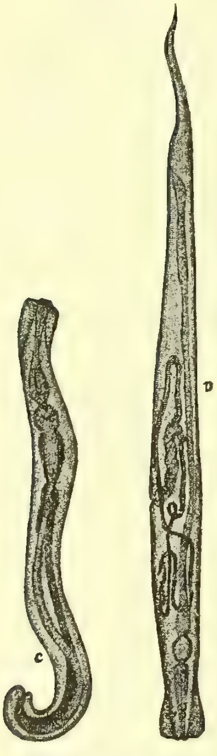
Prognosis is generally wholly favorable. Large numbers of round-worms may, however, prove serious, and migration to the biliary ducts, larynx, or nasal chambers is to be dreaded.

Treatment.—The most certain remedy in case of round-worm is santonin, which may be given to a child of five years in doses of a half to one grain, and to older persons in proportionately larger quantities. Several doses may be given during the day, and should be followed by a purgative after an interval of several hours. Very frequently calomel is given in combination with santonin in powders or in capsules, and this is perhaps the most efficient plan of treatment. Calomel alone is sometimes sufficient to effect the expulsion of the worms, but as a rule santonin is necessary to cause them to migrate to the large intestine, when the purgative completes their expulsion.

The compound infusion of spigelia and senna is a useful remedy, and is given in doses of a half drachm to a drachm until active purgation is induced. Oil of peppermint, oil of tansy, and other remedies have been employed, but are less efficient than santonin or spigelia, and some of them are not without danger of toxic action. Santonin, however, is also apt to occasion unpleasant symptoms in certain persons. The common manifestations are yellow vision, discoloration of the urine and the skin, delirium, and even convulsions.

ASCARIS MYSTAX, the round-worm of the cat and dog, has, according to Kelly, been observed in nine instances in man. It is distinguished by two wing-like crests on either side. The female is from three to four inches in length; the male, from one and a half to two and a half inches.

FIG. 73.



Oxyuris Vermicularis. C, male; D, female (after Heller) (magnified).

ASCARIS MARITIMA has been observed but once, and the female alone has been found. In this instance the worm was vomited by a young child in Greenland.

OXYURIS VERMICULARIS.—SYNONYMS.—Seat-worm; Pin-worm; Mawworm; Thread-worm.

The seat-worm is the commonest form of intestinal parasite of man, and is peculiar to him. It occurs in all parts of the world.

It is a rapidly-moving, wriggling organism. The female is about a half inch in length, the male about a fourth or third of an inch long. The posterior end of both tapers to a point. In the female the tail is elongated, while in the male it is short and curved ventrally, so as to form a somewhat hooked extremity. (See Fig. 73). The male is further provided with a chitinous spicule or penis. The anterior end of both is more blunt, and terminates in a head which has three papillæ surrounding the mouth. The eggs of Oxyuris are elliptical, about .05 mm. in length, and surrounded by a clear albuminous covering. The interior is of a granular nature.

The eggs are taken into the mouth by the host, and in the stomach lose their outer covering, so that the embryos are permitted to escape. These develop further in the small intestines, and in the lower part of the ileum the female becomes impregnated, when it passes into the cæcum. The males and young embryo are therefore found in the lower part of the small intestine, while the impregnated females occupy the cæcum, from which they pass downward when the eggs are ripe, and deposit the latter in the rectum, and are themselves discharged with the fæces.

The life of the individual worm is of limited duration, but the host is constantly reinfected by scratching at the anus and afterward carrying the eggs to the mouth with his uncleanly hands. Infants may be infected in a similar manner by their nurses. Children and women are more commonly affected than men. The disease is in general more common among those who are of unclean habits.

Symptomatology.—In some cases the presence of seat-worms is unsuspected, and gives rise to no symptoms for years, or indefinite symptoms may be present without the cause being discovered.

Itching at the anus is the most frequent symptom, and may give rise to great discomfort, especially at night, when the worms descend into the rectum. The child's sleep may be broken; grinding of the teeth, startings, and the like are common. The constant irritation of the worms may in a child of nervous temperament give rise to the same general disturbances of the nervous system as do lumbricoids.

Seat-worms are readily detected in the evacuations of infested children, but may also escape from the anus spontaneously, and are often found in the bed. In girls they may enter the vagina and induce vaginitis, and more rarely in boys gain access to the preputial folds, inducing balanitis. The irritation of the genitalia sometimes leads to onanism and general sexual excitability.

Itching about the nose and mouth is common, and leads to scratching and picking, with the attendant danger of reinfection by unclean fingers.

Large masses of seat-worms are sometimes discharged, and there may be a veritable verminous diarrhœa.

Diagnosis.—The detection of the worms is easy if the evacuations be examined.

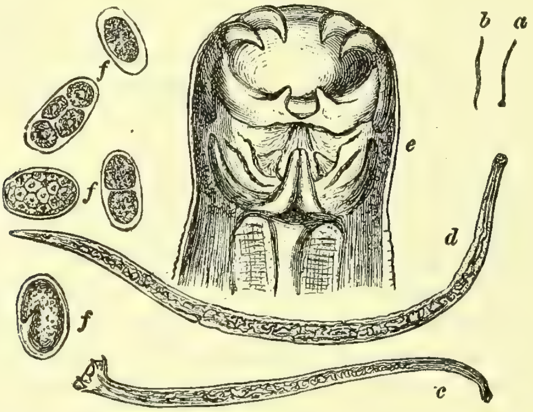
Prognosis.—Oxyuris is easily expelled, but it is so prolific and the danger of reinfection is so great that persons are sometimes infested for years.

Treatment.—Cleanliness on the part of nurses will often prevent the disease in children under their care. Remedies administered by the mouth are necessary to expel the worms from the small intestine. Magnesium sulphate, castor oil, or calomel may be used, and may alone suffice to remove the parasites. As a rule, however, rectal injections are also required. Immediately after a stool a large injection of warm water may be given to wash out the large intestine, and after this an enema containing a little vinegar or castile soap. Decoction of quassia, made by boiling an ounce of the chips in a pint of water, is a valuable remedy used as an enema. Olive oil containing a little oil of turpentine or carbolic acid is also useful in certain cases. The complete removal of oxyuris is difficult because they are apt to become lodged in the folds of the large intestine and in the cæcum and rapidly multiply. Repeated efforts must therefore be made, and the stools carefully examined for worms or eggs before the case is supposed to be cured.

ANCHYLOSTOMUM DUODENALE.—SYNONYMS.—Dochimus duodenalis; Strongylus duodenalis.

The anchylostomum is a cylindrical worm, the female having a length of six to nine lines, and the male about half or two-thirds the length of the female. The male is grayish or pinkish in color, and the female, owing to the blood which it has absorbed, is of a brownish or reddish hue. The anterior end in both sexes is tapering and somewhat recurved. The posterior end of the male is dilated into a sort of pouch, and its body is somewhat irregularly undulated. In the female the body is straight and the posterior extremity not enlarged. The head presents a capacious mouth armed with six hook-like teeth. The genital orifice of the female is situated behind the middle third of the ventral surface.

FIG. 74.



Anchylostomum Duodenale (after Jaksch). *a*, male (natural size); *b*, female (natural size); *c*, male (magnified); *d*, female (magnified); *e*, head (greatly magnified); *f*, eggs showing segmented condition of contents.

The number of anchylostoma found in the intestines may be inconsiderable, but, as a rule, they are very numerous, as many as fifteen hundred to three thousand having been counted in a single case. The females are more abundant than the male, the proportion, according to Leichtenstern, being 22 or 24 to 10.

The eggs of anchylostomum are easily distinguished. They are elliptical in shape, from .044 to .063 mm. in length and from .023 to .040 mm. in width. The shell is transparent, the contents brownish and constantly in the state of segmentation, by which feature alone the eggs are easily recognized. The further development of the egg and embryo takes place outside the body in water, where the embryo leads an independent existence for some time, after which it gains entrance to the stomach and intestines of man and undergoes complete development.

Etiology.—The anchylostomum does not pass one stage of its life in an intermediate host, but is developed directly, as was stated above, in the intestines of man. Anchylostomiasis is therefore observed in persons who are liable to drink of infected water or whose occupation requires the use of the hands in clay or earth containing the eggs or embryo.

The worm was first discovered and described in Italy by Dubini, and later Griesinger discovered that it is the cause of "Egyptian chlorosis." Perroncito found that the anæmia of the laborers at St. Gotthard's tunnel was also due to the same parasite, and during recent years anchylostomiasis has been seen among miners in various places in Europe. So far, no observations have been made in this country, but it is probable that the disease exists in some of the Southern States.

Seat and Morbid Anatomy.—The natural seat of the anchylostomum is the jejunum and the upper part of the ileum, but it is also found in the duodenum. By means of the sharp teeth arming the mouth it clings to the mucous membrane, and the head may penetrate this to the submucous coat, from which it sucks blood. The male, as a rule, abstracts much less than the female, and the latter generally presents a brownish color, which is imparted by the blood within. Ecchymotic spots are found upon the mucous surface where the parasites have fastened themselves, and there may be erosions or even small ulcerations. If the autopsy be made soon after death, the worms may be seen clinging to the surface of the gut and still in active motion. Anæmic degenerations of the heart, the liver, the kidneys, and of the glands of the stomach and intestines are observed in cases in which the patient has died of advancing anæmia.

Symptomatology.—The symptomatology of anchylostomiasis is practically the same as that of progressive pernicious anæmia. The severity of the symptoms depends to a certain extent upon the number of parasites present, since the general manifestations result from the abstraction of blood and from disturbances of the gastro-intestinal functions. Sometimes, however, severe cases are observed in which only a few worms are present in the intestines. For a variable time after infection the symptoms are indefinite and partake of the nature of gastro-intestinal disturbances. Heaviness or pain in the epigastric or umbilical regions, loss of appetite, nausea, vomiting, and alternating constipation and diarrhœa are first observed. Later the evidences of anæmia begin to declare themselves. The patient grows more and more pallid, palpitation of the heart and dyspnœa are frequent, and tinnitus aurium, vertigo, or syncopal attacks are usually experienced. The patient's strength deteriorates, copious sweating adds to his weakness, and there may be slight elevation of the evening temperature. The pulse is rapid and often irregular, and anæmic murmurs are heard over the heart and vessels. The blood is pale and watery, deficient in hæmoglobin, and the red corpuscles are greatly reduced in number. The number of red corpuscles is frequently below 2,000,000, and the hæmoglobin often under 40 per cent; but, as in pernicious anæmia, the percentage of hæmoglobin is usually somewhat greater than that of the red corpuscles.

The bowels are generally constipated, but there is sometimes obstinate diarrhœa, the stools being brownish or blackish in color, and sometimes distinctly hæmorrhagic. Microscopically, blood-corpuscles and Charcot-Leyden crystals are present in considerable number, and the eggs of the parasites are found in immense quantities. The worms themselves are rarely found unless anthelmintics have been administered.

In the final stages of the disease the patient becomes apathetic, emaciated, and finally dropsical. The serous cavities fill with fluid, urgent dyspnœa may be observed; there may be hæmoptysis, albuminuria, and severe nervous disturbances.

The disease may be acute, and lead to a fatal termination in a few weeks; as a rule, however, the duration is months, and with good food the patient may live for several years. Spontaneous discharge of the parasites with perfect recovery has never been observed.

Diagnosis.—Anchylostomiasis must be distinguished from idiopathic pernicious anæmia. In the latter retinal hæmorrhages are much more frequent, and the red corpuscles are more irregular in outline, than in anchylostomiasis. Nucleated red corpuscles are also more frequent in idiopathic pernicious anæmia. These points, however, are scarcely sufficient to warrant a distinction between the two conditions, and microscopic examination of the stools should therefore be made in every doubtful case. The eggs are so easily recognized by the segmented state of the contents, and are so numerous, that the diagnosis can always be made by examination of the stools.

Prognosis.—The termination is always fatal unless the condition is recognized and suitable treatment is instituted.

Treatment.—The prophylaxis is important in places where the disease is common and among laborers exposed to infection. The water used for drinking should, if possible, be clear running water, and should be carefully filtered and boiled. The workmen should be warned to wash their hands and faces before eating, and in general habits of cleanliness should be encouraged.

The most trustworthy remedy against anchylostomum is the oleo-resin of male fern. It should be given in large doses (one or two fluidrachms) in the morning, and after the same preparatory treatment as in the case of tape-worm. The patient must remain in bed during the day, and a purgative is given some hours after the oleo-resin. The treatment may require repetition when the presence of eggs in the stools indicates the failure of complete expulsion.

Thymol has been recommended in ascending doses, as much as a half drachm to two drachms being given in the day. Doliarine, the dried excretion of the Ficus doliaria, has been used in Brazil.

RARE FORMS OF ROUND-WORMS AND FLUKE-WORMS.

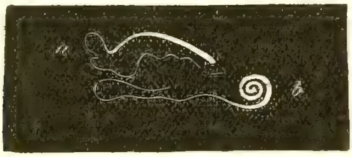
TRICOCEPHALUS DISPAR.—SYNONYMS.—Tricocephalus hominis; Whip-worm.

The whip-worm is a not infrequent parasite of the ileum and cæcum of man, but is usually present in small numbers, so that it gives rise to no symptoms and is unsuspected. The worms themselves are rarely discharged with the evacuations. It is an elongated thread-like creature, the female being about two inches in length and the male a third less. (Fig. 75). The posterior end is blunt and thick, and in the male is enrolled, while the anterior is attenuated in both sexes.

The eggs are of elliptical shape, about .050 to .055 mm. long, and have at each pole a knob-like prominence of clear, transparent structure. (Fig. 76). Infection by tricocephalus probably is direct, without any intermediate host.

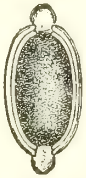
Ordinarily, no symptoms are observed, but if the worms are in large numbers pain in the abdomen, headache, and various reflex symptoms are said to occur.

Fig. 75.



Trichocephalus Dispar, natural size (after Heller).

Fig. 76.



Eggs of Tricocephalus Dispar, magnified.

Purgatives and the remedies employed for round-worm would probably suffice to remove this parasite.

Rhabdonema intestinale.—Synonyms.—Rhabditis intestinalis; Leptodera stercoralis vel intestinalis; Angulilula stercoralis.

This rare form of round-worm is found in Cochin-China, and has been seen occasionally in Europe and Brazil. It is about a half-line in length and attenuated anteriorly. It occurs in immense numbers in the jejunum, or more rarely in the ileum, and occasions violent diarrhœa or dysentery. Sometimes the worms enter the pancreatic or biliary ducts, and may lead to obstruction of these. Infection occurs by drinking stagnant water. The remedies used for other forms of round-worm may be employed, and the oleo-resin of male fern has seemed especially useful. Seifert has recently recommended thymol in doses of fifteen grains hourly, giving as much as three drachms in the day.

Amphistoma hominis.—But few observations have been made of this worm. It is a round-worm, about one-fourth of an inch long, and inhabits the cæcum and ascending colon. It was found by Lewis and others in India, and described by Cobbold.

Distomum heterophyes, a minute fluke-worm, about half a line in length, has been found but once. It occurred in the small intestine.

Distomum crassum is the largest of the fluke-worms infesting man. It is elliptical in outline, thick and smooth, and varies in length from one to three inches. It is found in the duodenum, and has been observed a number of times in China and India.

Distomum hæmatobium has been found occasionally in the mucous membrane of the intestines, where it may occasion destructive ulceration.

Pseudo-parasites.

Various materials passed with the stools may be mistaken for parasites, and occasionally larval forms of insects are found, whose presence of course depends upon accidental circumstances. Very frequently in cases of mucous colitis the casts or tubes discharged are regarded as portions of tape-worm. Similarly, undigested vegetable tissues, such as portions of onions, are thought by patients to be parasites. Recently I have seen two cases of diarrhœa in which the juice-cells of oranges were passed in considerable numbers.

Larvæ of various flies may be passed in large numbers. In a case recorded by Wacker two litres of larvæ of the fly, Anthomya cuniculina, were discharged from the bowel of a young man. In a case of my own the patient reported that there were great masses of the larvæ. Pain in the abdomen, diarrhœa, and emaciation are sometimes noted in these cases.

DISEASES OF THE PERITONEUM.

BY REGINALD H. FITZ.

INTRAPERITONEAL HÆMORRHAGE; HÆMATOCELE; HÆMOPERITONEUM.

HÆMORRHAGE into the peritoneal cavity is either circumscribed (hæmatocele) or diffuse (hæmoperitoneum).

In hæmatocele the bleeding takes place from ruptured blood-vessels in the ovaries, uterus, tubes, broad ligaments, and pelvic peritoneum, and its extent is limited by pre-existing adhesions. In hæmoperitoneum the bleeding may proceed from vessels in any part of the abdominal wall or its contents, and its progress is unobstructed, owing to the absence of limiting adhesions. The hæmoperitoneum may result from a hæmatocele, but it is doubtful whether a hæmatocele may result from a hæmorrhage into the normal peritoneal cavity (Veit).

Etiology.—The causes of hæmatocele are local and exciting. The former are to be found in varicose vessels near the surface of the ovary, especially in the walls of dilated Graafian follicles. Such vessels may first break into the follicles or ovarian stroma, producing a hæmatoma of the ovary which may later open into the peritoneal cavity. The rupture of vessels within the uterine wall is unlikely to produce a hæmatocele except in the case of early pregnancy within a rudimentary horn, the pathological significance of which is essentially the same as that of tubal pregnancy. Rupture of a dilated Fallopian tube is the chief local cause of hæmatocele, the dilatation being due to ectopic gestation. Dilatation of the tubes from the obstructed outflow of blood, as in hæmosalpinx, is rarely followed by hæmatocele, since the wall of the tube is symmetrically dilated and thickened, and the fimbriated end is tightly closed by fibrous adhesions.

The possibility is rare, but may be admitted, of the occurrence of a hæmatocele from the escape of menstrual blood through the fimbriated end of a normal tube or from the open mouth of one cut across in the removal of a pelvic tumor.

The frequent occurrence of varicose veins in the broad ligament suggests that their rupture may result in a hæmatocele. The rupture may also take place between the peritoneal folds which form this ligament, producing a hæmatoma of the broad ligament, which may subsequently break into the peritoneal cavity, like the ovarian hæmatoma.

Finally, especial importance is to be attached to a hæmorrhagic pelvic peritonitis as a cause of hæmatocele. This condition is analogous to the hæmorrhagic inflammation of the inner surface of the dura mater, and, like it, may result in a serous accumulation of fluid beneath the new-formed membrane produced by the inflammation. The blood-vessels in this membrane are thin-walled, sometimes large, and their rupture may result in extensive hæmorrhage.

The two, chief, local causes of hæmatocele are almost universally admitted to be a ruptured tubal pregnancy and a hæmorrhagic peritonitis. The former was first advocated as the chief cause by Gallard in 1855.

The exciting causes may be found in blows, falls, or strains, or in congestions from exposure to cold. They are more likely to arise from physiological conditions, as the growth of the fœtus and its villi, with a progressive thinning of the wall of the tube. Physiological exciting causes are also to be found in the congestion of the pelvic blood-vessels at the menstrual period and during sexual excitement, which may, at times, be combined.

A certain importance in etiology may be attached to the presence of scurvy, purpura, and hæmophilia, the infectious diseases, and phosphorus-poisoning.

The causes of hæmoperitoneum are traumatic, as stabs and bullet-wounds, which cut or tear the blood-vessels of the abdominal wall or viscera. In this connection allusion may be made to fatal hæmoperitoneum following the operation of tapping for ascites. Also traumatic are the lacerations of the liver, spleen, and kidneys and the lacerations and perforations of the blood-vessels of the stomach and intestines. Rupture of the pregnant uterus or Fallopian tube is a partly physiological, partly pathological, cause, while ruptured aneurisms of the aorta and its larger abdominal branches or of the omental and mesenteric arteries are wholly pathological. Under the latter head are to be included the intraperitoneal hæmorrhages from the rupture of vessels in malignant tumors of the liver, pancreas, and ovary.

The most important of all causes of hæmoperitoneum are the remedial causes—viz. a ruptured tubal or uterine pregnancy and an aneurism, especially of the omental or mesenteric artery.

Morbid Anatomy.—The hæmatocele shows itself as a rounded tumor, which may be larger than a child's head, though usually nearer the size of an orange, and may extend from the pelvis into the abdominal cavity. At the outset it is usually retro-uterine, but with its increase in size, or even at the outset, it may occupy the vesico-uterine space. Hence the uterus lies near the pubes unless the hæmatocele be antero-uterine. It may become so large from progressive bleeding as to be walled in by adhesions between the intestines and the peritoneum of the abdominal walls.

The periphery of the tumor is composed of clotted blood, old or fresh, adherent to the thickened peritoneum and entangled between fibrous adhesions. The centre contains darker, softer, even liquid blood, that last extravasated, which may be crossed by fibrous adhesions. In rare instances the hæmatocele may project into the peritoneal cavity as a rounded mass of clotted blood not walled in by adhesions.

In hæmoperitoneum, where there is extensive hæmorrhage, the pelvis and dependent portions of the abdominal cavity contain soft, dark clots lying upon and between the intestinal coils, and forming a thin layer between them and the anterior abdominal wall, usually extending from below upward.

Symptoms.—Intraperitoneal hæmorrhage produces a sudden oligæmia. According to its degree, there is unexpected exhaustion and debility, progressive pallor of the face, increasing rapidity and diminishing strength of the pulse; or the patient becomes rapidly collapsed, the face is pinched and sunken, the expression anxious and fearful, the extremities cold, the skin clammy, the respiration deep and sighing, the voice hollow and husky, the pulse almost imperceptible.

Pain is an incipient symptom of no extreme severity, and soon yields to the graver signs. It is chiefly important as directing attention to the probable source and seat of the hæmorrhage. In tubal pregnancy occasional twinges of sharp pain may for days precede the actual rupture of the tube. In such cases a delicate fibrinous membrane indicates the peritonitic origin of the pain, while the discharge of uterine decidua calls attention to the existence of ectopic gestation.

The physical examination will give but little aid in determining the seat or extent of the hæmorrhage unless the latter be limited by adhesions. The thickness of the clot is usually insufficient to produce any marked dulness on percussion, and dulness from intestinal contents may be present with little or no hæmorrhage. The resistance of the free blood is no greater than that of the intestinal coils with their liquid contents.

If, on the contrary, the hæmorrhage be limited by adhesions, a tumor, the hæmatocele, is formed, and is usually to be recognized within twenty-four hours after the onset of the hæmorrhage. The symptoms are essentially those of pressure upon the bladder, rectum, or pelvic plexus of nerves, and are frequent micturition, painful dejections, pain or abnormal nervous sensations in the legs.

Vaginal examination shows that the wall of the vagina is depressed from behind or in front by an elastic tumor; the uterus is high in the pelvis and lies close behind the symphysis, or its fundus is near the sacral promontory, according to the retro- or antero-uterine seat of the tumor. Bimanual examination, unless in anæsthesia, gives but little additional information at this early stage, owing to the sensitiveness and tension of the abdominal wall.

If the patient survive, the symptoms of a mild, localized peritonitis now become apparent. Slight fever preceded by chilliness, hypogastric and vaginal tenderness, and moderate tympany are present. The fever usually lasts but a few days; the tenderness may remain for weeks. The tumor tends to become firmer, smaller, and in the course of months is eventually represented merely by a diffuse induration. In other instances it becomes increased in size, with a recurrence of all the symptoms of the original acute attack. The more frequent these attacks, the graver becomes the condition of the patient and the more likely the occurrence of perforation. This event may also take place in the case of the larger hæmatoceles, without any necessary recurrence of the hæmorrhage.

The perforation most often takes place into the rectum, and may be forewarned by a preceding irritability of this portion of the intestine, shown by frequent mucous discharges and tenesmus. A rectal examination may reveal a soft spot in the, elsewhere, hard wall of the tumor—an indication of the part at which the discharge is to take place. The temporary relief following the evacuation of the hæmatocele into the rectum may become permanent, with the eventual disappearance of the hæmatocele. On the other hand, the admission of the rectal contents may result in a putrefaction of the contents of the hæmatocele and a gangrene of its wall, ending in a peritonitis which may prove fatal.

Perforation may also take place into some other part of the intestinal canal, and permanent fistulæ may be established, through the hæmatocele, between the ileum or cæcum and the rectum. Perforation into the vagina may occur, though less frequently than into the rectum, and perforation into the bladder is extremely rare. Perforation into the vagina may be followed by relief, although the same unfortunate results may arise as in the case of rectal perforation. Perforation into the bladder leads to a new source of danger—viz. pyelo-nephritis.

Diagnosis.—The diagnosis of intraperitoneal hæmorrhage is either clear, as in the traumatic varieties, or is based upon the occurrence of a sudden, unexpected collapse in an apparently healthy person, accompanied with pelvic or abdominal pain, and progressing rather with symptoms of anæmia than with those of peritonitis.

A gastric, intestinal, uterine, or urinary origin of the hæmorrhage may be eliminated by the absence of symptoms of previous disease of the organs concerned and of pregnancy, as well as by the absence of blood in the vomit, stools, urine, or in the vagina.

An intestinal source of so considerable a hæmorrhage is usually accompanied by pain in the region of the duodenum, or by symptoms of typhoid fever, or by evidence of a chronic fibrous hepatitis. Such extreme hæmorrhage from tumors of the liver, pancreas, or ovaries occurs only in the later stages of these affections, which have already been manifested by characteristic symptoms.

A similar collapse may follow the rupture of an ovarian cyst; hence the importance of seeking for a tumor in addition to recognizing the symptoms of collapse and anæmia. The search for a tumor is further important, since hæmorrhage may take place into an ovarian cyst with a twisted pedicle, the symptoms being the same as those resulting from hæmorrhage into the abdominal cavity. An ovarian cystoma large enough to produce such grave symptoms by the rupture of a cyst is likely to have been discovered before the rupture. One with a twisted pedicle may only be appreciated after the symptoms of hæmorrhage have taken place. It is then readily recognized, as a sudden, rapid enlargement is likely to follow the twisting of the pedicle.

If a hæmatocele results from the hæmorrhage, the tumor may be confounded with a circumscribed perimetritis, which it may closely resemble in symptoms, seat, and physical characteristics, often requiring the use of an aspirator for the differential diagnosis. In less extreme cases the symptoms of a perimetritis are not so violent and rapid; the anæmia is lacking and the tumor is less tense.

Retroflexion of a pregnant uterus may closely resemble a hæmatocele. It is to be eliminated by the history of pregnancy, probable urinary retention, and a dilated bladder, to be shown by the passage of a catheter or sound.

. Hæmatocele may be simulated by pregnancy or retained menses in a rudimentary horn. The history, again, becomes important, as suggesting pregnancy with periodical uterine pains and absent flow at the menstrual period. The tumor is rather lateral, and the os becomes dilated, crescentic, and is a part of the tumor.

Uterine fibromyomata or small ovarian tumors may be mistaken for the hæmatocele, but lack the history.

A hæmatoma of the broad ligament also lacks the history of shock and peritonitis, and the tumor is lateral, pushing the uterus sideways.

Prognosis.—Small intraperitoneal hæmorrhages are rarely absorbed. Large hæmorrhages into the normal peritoneal cavity prove fatal in the course of a few hours or in a day or two. The hæmatocele, under favorable circumstances, is gradually absorbed, although more or less prolonged discomfort may result from the associated chronic pelvic adhesions, obliterated tubes, and dislocated organs. Dysmenorrhœa, sterility, and chronic invalidism are not unlikely results.

With perforation come the dangers of gangrene and septicæmia, with or without general peritonitis or pyelo-nephritis.

Treatment.—Cases of intraperitoneal hæmorrhage are to be treated either surgically or expectantly. Immediate laparotomy is demanded when the anæmic symptoms persist, especially if no tumor be found or a tumor be present with a history of ectopic gestation.

If a tumor be present and the patient's condition warrant it, the laparotomy may be postponed for a few days in the case of an ovarian tumor, that the patient may rally from the shock of the ruptured cyst or twisted pedicle. If a tumor follow the symptoms of hæmorrhage, it is either a hæmatocele or a hæmatoma, and, as a rule, the blood becomes absorbed without serious disturbance. In this event the patient should be treated by applications of ice to the abdomen, rectal or vaginal enemata of cold water, and opiates sufficient to relieve pain.

The strictest rest should be enforced. The bladder should be catheterized if micturition be painful or difficult, and saline drinks should be ordered in sufficient quantity or strength to produce soft or fluid stools.

If, as stated by Gusserow, the hæmatocele be so large as to be mechanically disturbing, or if absorption of the extravasated blood be at a standstill, or if the circumstances of the patient do not permit slow absorption or prolonged rest after absorption, or if the contents of the hæmatocele become putrid, as indicated by symptoms of septicæmia—it should be opened, emptied, and drained without delay.

Zweifel gives the following statement of the results of treatment: Of 144 cases treated expectantly, there was a mortality of 16.6 per cent.; of 66 cases treated by puncture, 15.1 per cent. ended fatally; and of 30 cases treated by incision, there was a mortality of 10 per cent. Future experience is likely to show a smaller mortality from the last method.

HYDROPERITONEUM; ASCITES; ABDOMINAL DROPSY.

Ascites, the term in ordinary use, is applied to the accumulation of transuded serous fluid into the peritoneal cavity. It is a symptom occurring in a variety of diseases.

Etiology.—Serum readily transudes through the normal peritoneum, passing into the cavity through the walls of the radicles of the portal vein, and being absorbed from it largely through the lymphatics. Hence obstructions to the flow through either of these channels will promote transudation from the former, and will check absorption through the latter. The mechanical varieties of ascites thus arise. If the endothelial covering of the peritoneum be abnormal, transudation is checked, especially absorption by the lymphatics; ascites thus produced is called hydræmic or cachectic.

The mechanical causes of abdominal dropsy are general or local. General causes are to be found in obstruction to the passage of blood through the heart, especially from uncompensated valvular disease, cardiac weakness, emphysema of the lungs, and fibrous pneumonia. Local causes are to be found in obstruction to the passage of blood through the liver, as in fibrous hepatitis, malignant tumor of the liver, obstruction of the trunk of the portal vein by thrombosis, or pressure upon it by tumors of the pancreas or enlarged lymphatic glands, and constriction of it by peritonitic indurations and adhesions. The local obstruction to the outflow of blood and lymph may result from the pressure of tumors upon the lymph-vessels and hepatic vein or from the pressure of a tumor upon the inferior vena cava.

Hydræmic or cachectic ascites is considered to be the result of the disturbed nutrition of the peritoneum in wasting diseases, amyloid degeneration, nephritis, and in chronic malarial poisoning. The accumulated transudation so often associated with tuberculosis and cancer of the peritoneum is more allied to peritonitic exudation than to dropsical effusion.

The most frequent cause of the graver varieties of ascites is chronic fibrous hepatitis.

Although ascites is usually acquired and an affection of adult life, it may be found in children, and may exist at birth, interfering with the delivery of the infant and requiring removal of the fluid that this may take place.

Morbid Anatomy.—The peritoneum may show little or no alteration, or it may be slightly thinned and opaque, especially in patches, which are usually superficial. The subperitoneal fibrous tissue may also be thickened and indented. Such changes are more likely to be found in cases where abdominal puncture (tapping) has been necessary, and are rather complications than essentials of the ascites.

The accumulated transuded fluid, the dropsical effusion, is pale yellow, with a greenish tint, usually clear, though sometimes opalescent from some unusual, probably albuminoid, constituent. The color may be darker green from the presence of biliary coloring matter, or may have a reddish or brownish tint from the presence of blood-pigment. It is of watery consistency, though sometimes viscid, alkaline, or neutral, and has a specific gravity of 1010–1015. When cancer of the liver is the cause of the ascites, the specific gravity may be 1023, and the color is usually dark from the presence of bile- or blood-pigment. Its chemical characteristics are essentially those of blood-serum. In uræmia it may contain large quantities of urea. The percentage of albumin present varies between .3 per cent., or less, in hydræmic ascites, and 6 per cent. in old transudation with marked abdominal distension. Reuss gives the following formula for determining the percentage of albumin, based upon the determination of the specific gravity of the fluid, which is readily obtained by means of the urinometer. The fluid should be allowed to stand for twelve hours before determining its specific gravity, that its temperature may be that of the room and that its gases may disappear. The formula is as follows:

$$E = \tfrac{3}{8}\,(S-1000)-28.$$

E is the desired percentage of albumin, and S represents the specific gravity of the fluid. Errors are in the vicinity of .25 per cent.

It may prove of diagnostic importance to determine the percentage of albumin in the peritoneal fluid, since less than 1.5 per cent. of albumin is indicative of ascites, while more than 2.5 per cent. is suggestive of peritonitis. The following table shows at a glance the diagnostic value of the determination of the specific gravity of the fluid by means of the urinometer, and is a modification of that prepared by Runeberg:

Sp. gr.	Percentage of Albumin.		Sp. gr.	Percentage of Albumin.	
1008	0.2	Hydræmic ascites.	1017	3.6	Cancerous peritonitis.
1009	0.6		1018	4.0	
1010	1.0	Portal obstruction.	1019	4.3	
1011	1.3		1020	4.7	
1012	1.7	General venous obstruction.	1021	5.1	
1013	2.1		1022	5.5	
1014	2.5	Peritonitis.	1023	5.8	
1015	2.8		1024	6.2	
1016	3.2				

When the fluid is allowed to stand, a delicate fibrinous clot forms. There is but little sediment. This is composed of leucocytes, granular or fatty corpuscles, endothelium, vacuolated or granular red blood-corpuscles, and cholesterin. In the sediment of the fluid from general venous obstruction the red blood-corpuscles are numerous.

Ascitic fluid sometimes presents an opaque white color, resembling that of milk. This is due to the admixture of chyle, chylous ascites; or of fat, adipose ascites. In the former the fat is molecular; in the latter, it is in large and small drops.

Chylous ascites is caused by injury to the lacteals from wounds, ulcers, or rupture and from the presence in them of filariæ. It may be caused by thrombosis of the thoracic duct, or by obliteration of the duct by scars, or by mesenteric induration along the attached border. Thrombosis of the subclavian vein at the point of entrance of the thoracic duct has been proven a cause. When the obstruction is near the subclavian vein a milky fluid may also be found in the left pleural cavity.

If the fluid be allowed to stand, a creamy layer rises to the top and a gelatinous clot forms at the bottom. The specific gravity is high, 1023, and the percentage of albumin large, from 3 to 5.5 per cent. The sediment contains red blood-corpuscles, endothelium, and molecules, which fuse into fat-drops when warmed.

In adipose ascites the fluid owes its milky appearance to the presence of fat. The fat is the result of the fatty degeneration of cells which are present in the fluid. These cells are usually the product of a cancer or tuberculosis of the peritoneum or of a chronic peritonitis.

Symptoms.—The symptoms of ascites are attributable to the exudation which presses upon the abdominal walls and contents and produces a mechanical disturbance, the degree of the latter of which varies in accordance with the quantity of fluid present. Such disturbance is usually gradual and progressive, although F. C. Shattuck reports a case where the fluid accumulated to such an extent in five days that tapping became necessary for the relief of the patient.

The symptoms resulting from the distension are a sense of fulness or weight, which eventually becomes one of pressure. The resulting discomfort is rather distress than pain. This pressure causes the abdominal walls to become distended, the liver is forced into the thorax, and the heart and lungs are pushed upward and backward. The apex of the former may be found in the third intercostal space, and the upper border of hepatic dulness may lie in the vicinity of the third rib. The patient may be unable to lie down, and the legs become œdematous. Breathing becomes rapid and labored in consequence of the pressure upon the diaphragm, lungs, and heart. Vomiting takes place easily, and the bowels are apt to be constipated. Micturition is frequent, but the urine passed is small in quantity, high-colored, with high specific gravity and a brick-dust sediment. It contains a trace of albumin, and presents the characteristics of a chronic passive congestion of the kidneys.

The distended abdomen frequently contrasts markedly with the emaciation elsewhere. When the patient lies on his back the abdomen is flattened in the epigastric region and bulges in the flanks. In the upright position the bulging is toward the front. In extreme distension the belly is rounded. The surface of the abdominal wall is smooth and shining, and the skin is dry, seamed with pink or red streaks as in pregnancy. Its veins are enlarged and distended, and there may be a reversal of the current, and in extreme hepatic ascites they may radiate outward from the navel. Blood from the portal radicles may thus pass through dilated veins in the round ligament into those of the abdominal wall, and then enter the heart without passing through the liver. Another outlet for the intestinal blood is to be found in the dilated œsophageal veins lying between the branches of the azygos and portal veins. The navel may be obliterated or it may protrude as a soft, rounded, and translucent tumor.

Percussion of the distended abdomen gives resonance over the floating stomach and intestines. In the dorsal position these usually lie toward the epigastrium. The line of dulness is generally curved, owing to the higher position of the fluid at the sides than at the median line. With a change to the lateral position the line of dulness shifts to the dependent part of the abdomen, as the intestines float on the shifting surface of the fluid.

This characteristic area of dulness may be modified when the intestines contain fluid instead of gas or are prevented from floating on the surface of the fluid, either because the quantity is enormous or because the mesentery is shortened or adhesions are present. If intestinal resonance is then to be recognized, it may be found at the sides instead of in front, and may not change with a change of position.

Fluctuation of the abdominal fluid is ascertained by the transmission of a wave from one part of the abdomen to another by tapping with the finger of one hand and receiving the impression with that of the other. The wave is most readily perceived near the top of the dull area, and may be seen if the abdomen be but moderately tense. If the abdominal walls are excessively fat or œdematous, they may transmit a wave whose progress is to be checked by the intervening pressure of the ulnar edge of the hand or the edge of a book. If the tension of the abdominal wall be very great or very slight, there may be no perceptible wave.

The **course** and **duration** of ascites depend largely upon the cause. In general, the condition is chronic, and, if there be a diminution in the quantity of fluid, the relief is but temporary and recurrence takes place. A discharge of the fluid may take place through the navel or into the rectum, and the fluid may disappear spontaneously in hepatic ascites as the abdominal veins dilate.

Diagnosis.—The diagnosis of ascites depends upon the recognition of the presence of fluid in the abdominal cavity and an appreciation of its cause. The presence of fluid is indicated by the shape of the distended abdomen, the limitation of dulness to the dependent parts, and the recognition of fluctuation. It is not always easy to decide that the fluid is free, especially in the female. A large unilocular ovarian cystoma or a parovarian cyst may simulate ascites. The former may permit a displacement of the intestines on change of position, thus causing an alteration in the level of dulness. This variation is less likely in the case of the cyst than in the case of ascites. The upper border of this line is likely to be convex upward in the case of the cyst, concave in case of the free fluid. In ascites the wave is more likely to be transmitted into Douglass's fossa, and the vault of the vagina is often obliterated. The uterus is more freely movable in the case of ascites than when a pelvic tumor is present. The possible coexistence of a cyst and ascites is to be borne in mind. Although a pregnant uterus or a uterine fibro-cyst or dropsy of the amnion may be confounded with ascites, auscultation may eliminate the first, the passage of a sound the second, and the recurring watery or bloody vaginal discharge with an elongated uterine cavity the last.

In both sexes ascites may be simulated by a distended bladder, a dilated stomach or intestine. The passage of a catheter will empty the distended bladder. The dilated stomach may be so distended with liquid contents as to extend to the pelvis and to transmit a visible wave across the abdomen. The progress of the tumor is distinctly from above downward, and the history of the case points to early disturbance of the functions of the stomach. Litten, who calls attention to the possibility of mistaking the dilated stomach for ascites, also mentions an enormous dilatation of the ileum from tuberculous strictures which closely simulated ascites from the existence of an extreme distension of the abdomen, dulness on percussion, a change in the area of resonance, and fluctuation.

It may be impossible to discriminate between the distended abdomen from chronic or tuberculous peritonitis and that from ascites, especially when there are no adhesions. In such cases the history of the case is important. In the peritonitic cases pain and an elevated temperature are more likely to be present. Evidences of tuberculosis may be found elsewhere, or the thickened peritoneum, omentum, or mesentery may be felt. The absence of evidence of disease of the liver, heart, and kidneys is also of importance in the exclusion of ascites.

The examination of the fluid secured by puncture may be of great assistance in diagnosis in cases of doubt.

Typical ovarian fluid is viscid, semi-opaque, has a specific gravity of 1018–1055, and contains much albumin. It does not clot, and contains cylindrical or globular epithelium, often in a condition of hyaline or fatty degeneration. The fluid from peritonitis is likely to have a specific gravity above 1014, and a percentage of albumin above 2.5 per cent.

The pale watery fluid with a specific gravity of 1005 and a trace of albumin is sufficiently characteristic of the parovarian cyst, while the fluid from a fibro-cyst of the uterus contains a more abundant fibrinous clot.

The examination of the abdomen after removal of the fluid may clear up the diagnosis of the disease by disclosing an atrophied liver, an enlarged spleen, or an abdominal tumor.

Prognosis.—The prognosis of ascites is always grave in consequence of the usual progressive nature of the disease which causes it, and the occurrence of this symptom often represents the approach of a fatal termination. It is the more serious the greater the quantity of fluid, the longer it persists, and the more rapidly it returns after removal.

The less the evidence of disease of the organs in which ascites is a symptom, the more favorable is the prognosis. Cases occur when, after removal of the fluid, there has been no return. Laparotomies have revealed a benignant tumor, the removal of which has cured the ascites, and life has persisted for fifteen years, during which time the abdominal fluid has been withdrawn more than eight hundred times (Lecanu). The possibility of error in diagnosis in such cases is exhibited by a case which came under my observation where the abdomen was tapped seventy times in twenty years, two pailfuls at a time, and a large monocyst arising from the pelvis proved to be the source of the fluid, the peritoneum presenting a normal appearance.

Treatment.—The treatment of ascites consists in attempts to remove the fluid from the peritoneal cavity through the intestines, kidneys, skin, or abdominal wall. For this purpose cathartics, diuretics, diaphoretics, electricity, and puncture may be employed. The benefits to be derived from medicinal treatment are, as a rule, but slight and temporary, since the causes of ascites are usually persistent and progressive. An objection to the prolonged or continuous use of eliminative drugs comes from the digestive disturbances they produce.

Among the cathartics, the stronger mineral waters may be daily used, as Hunyadi or Friedrichshall water. Sulphate of magnesia has long been a favorite remedy in daily doses of one or two teaspoonfuls in the morning in a tumbler of water, or it may be taken in combination with calomel and jalap, a half ounce of the first-mentioned salt with 10 grains of each of the latter drugs. This powder should be taken before breakfast in a half-tumblerful of water, and may be repeated at intervals of a day or two as the effect is beneficial or weakening.

If the patient be of fairly good strength, more active purgation results from diminishing the quantity of water to a little more than sufficient to dissolve the magnesium sulphate. A combination of jalap and bitartrate of potash, as in the compound jalap powder, in teaspoonful doses, is often useful and less disagreeable than the combination of jalap and sulphate of magnesia. All saline hydragogue cathartics are better given in concentrated solutions before breakfast, when the stomach is empty, that their action may be more irritating, and purgation take place at a convenient hour.

Bitartrate of potash is a convenient diuretic, and may be taken in the form of a drink, a teaspoonful to the pint of sweetened water, at pleasure, through the day. Of late years the use of calomel and digitalis has been resumed, the diuretic action of the digitalis being intensified by the combination with calomel, while frequently-repeated small doses of calomel are recognized to have diuretic properties. Although the beneficial effects of calomel and of calomel and digitalis in ascites are not as striking as in the general dropsy of cardiac disease, there are conspicuous instances of marked relief from their use. The quantity of urine has been increased from one pint to five pints in the course of twenty-four hours. A powder of 3 grains of calomel and 1 or 2 grains of powdered digitalis-leaves may be given three times daily until the mouth begins to feel sore or diarrhœa supervenes. The addition of one-third of a grain of powdered opium may be of benefit in delaying the onset of the injurious effects of the calomel. If they occur, the medicine should be discontinued, to be resumed, if desired, after an interval of several days. The diuretic action usually begins within two or three days, and may continue for a few days after the calomel and digitalis are stopped.

Faradization of the abdominal muscles has been employed in the treatment of ascites. The success is determined by an increased flow of urine and a diminution of the abdominal enlargement. A strong current should be used for about ten or fifteen minutes daily.

The only diaphoretics worth mentioning are pilocarpine subcutaneously and heat. The former is capable of producing harm by weakening the action of the heart, thus favoring the occurrence of œdema of the lungs. The advantages are so slight compared with the possible risk as to make its use undesirable. Heat is one of the most useful diaphoretics, and may be used in moist or dry form. In case the patient is able to sit up, he may be placed in an ordinary cane-seated or wooden chair, and a blanket so fastened about the neck as to cover the patient and the chair, a small space being left below for the admission of air. A lighted lamp, with a small flame, may be placed beneath the chair. The patient may remain in this hot-air bath for some ten or fifteen minutes unless he feels faint or complains of headache. The vapor-bath may be provided for in the same way, a nursery lamp being substituted for the illuminating lamp, that damp hot air may be produced by the steam from the boiling water. Steam may be introduced directly beneath the chair or bed-clothes through a tin tube of desirable length soldered to the cover of any metallic vessel of the capacity of two quarts or more, the water in which may be heated to boiling by means of an alcohol or gas flame. Lesser degrees of sweating may be readily induced by placing beneath the blankets covering the patient bottles or rubber bags containing hot water and covered with damp cloths. Heated bricks and plates are familiar means of producing a sweat.

Eventually, as a rule, tapping becomes necessary to relieve the distress from the abdominal distention. The necessity for its use often means a late stage in the disease which has produced the ascites, and in most cases it is to be regarded merely as a means of adding to the immediate comfort of the patient, as the fluid quickly returns.

In cases of curable ascites the fluid may not return after a few tappings, and life may be prolonged for years. Removal of the fluid often produces a sensation of faintness, which the sooner occurs the more rapid the removal of the fluid. Fatal peritonitis has followed the use of unclean trocars, and fatal hæmorrhage has resulted from the puncture of arteries in the abdominal wall.

The patient may sit up, if able, resting his hand and head upon a chair in front. If unable to leave the bed, he may be placed in a semi-prone position on his back, near the edge of the bed. The puncture should be made in the median line midway between the symphysis and navel, or on either side midway between the symphysis and the antero-superior spine. The skin should first be cleansed by scrubbing with soap and water, then with a 5 per cent. carbolic solution or one of corrosive sublimate 1 : 3000. The puncture should be made with a straight trocar, one-eighth of an inch or less in diameter, previously cleaned by heat, and it should be made evident by previous percussion that an intestinal coil does not lie directly beneath the place where the patient is to be tapped. Pressure should be applied by a swathe to the abdominal walls as the fluid escapes. If the mouth of the trocar becomes obstructed by floating intestine, a change in the direction of the former or the insertion of an aseptic probe will remove the obstruction. Fluid may be allowed to escape until the flow ceases, in which case the trocar is to be removed. A strip of adhesive plaster may be placed over the opening, some oozing from which is frequent and beneficial : when necessary the opening may be closed by a suture. Antiseptic gauze and absorbent cotton may be placed on the plaster, and the abdomen tightly swathed. The patient should then remain in bed for a few days, after which time he may be allowed to sit up.

The quantity of fluid removed at one puncture may be as high as ten quarts, and the fluid may be expected to so reaccumulate in the course of three weeks that a further tapping becomes necessary. After the removal of the fluid it may appear, especially in women, that an abdominal tumor is present. Such tumors may be the cause of the ascites, and their removal by laparotomy may effect a cure.

Inflammation of the Peritoneum; Peritonitis.

Inflammation of the peritoneum is acute or chronic, circumscribed or diffuse, primary or secondary, and may be considered under the one or the other of these headings. Of chiefest clinical importance are the acute and chronic varieties, which are often not to be sharply differentiated.

Etiology.—It has been found convenient in the consideration of the causes of peritonitis to discriminate between the primary and secondary varieties of inflammation of the peritoneum. In the former the cause is obscure, hence it is called spontaneous or idiopathic; while the secondary variety is the result of obvious causes to be found in parts covered by peritoneum. The value of such a distinction is remote, since most cases of peritonitis belong to the secondary variety. In rare instances, however, a peritonitis develops suddenly, rapidly, with severe symptoms and important lesions, after exposure to cold; the patient dies, and no other lesions than those of the peritoneum are found. Such cases are sometimes called rheumatic, and the occasional development of a peritonitis during the course of an acute articular rheumatism is to be met with.

Although cases sometimes occur where a primary source of the peritonitis is undiscoverable, the tendency of pathological researches is to offer a reasonable explanation of the secondary nature of these instances.

The importance of bacteria in the etiology of peritonitis is well recognized, and the search for bacteria in the products of peritoneal inflammation shows that no single variety is alone concerned in the inflammatory process. Not only are pyogenic bacteria found in the exudation, but the presence of other varieties also has repeatedly been shown. The colon bacillus has been found with such frequency, especially in cases of intestinal origin, that its presence in the exudation has even been regarded as of diagnostic importance in determining the place of origin of the peritonitis. Experiments on animals, especially those of Grawitz, Orth, and Reichel, suggest that the presence of bacteria alone does not suffice to explain the origin of the peritonitis, but that the association of other factors is necessary, as traumatism, chemical and mechanical irritants, pre-existing disease (ascites, heart disease, fibrous hepatitis), which interfere with the absorbing powers of the peritoneum.

A distinction may thus be drawn between a simple, bland peritonitis and an infectious, bacterial, septic peritonitis. The former may be caused by trauma alone, the outpoured contents of a ruptured ovarian cyst, or by the twisting of the pedicle of a pedunculate tumor, as a cystoma or a fibro-myoma.

The septic variety, on the contrary, is that caused by the association of bacterial infection, traumatic or chemical irritation, and a peritoneal lesion. The presence of solid material absorbed with difficulty, as fæces and blood-clots, favors the bacterial influence. Such bacteria may be admitted directly into the peritoneal cavity from parts covered by peritoneum and communicating with the surfaces of the body, as the gastro-intestinal canal and the digestive glands, the genito-urinary apparatus, and subcutaneous abdominal wall. They may enter through open-mouthed Fallopian tubes or obtain passage through an adherent diaphragm from the pulmonary surface. It is probable that the infectious bacteria may reach the peritoneal cavity from more or less remote points through the circulating blood or lymph. The occurrence of peritonitis in diphtheria, erysipelas, acute articular rheumatism, pneumonia, and cerebrospinal meningitis may be thus explained in the absence of obvious, immediate, exciting causes.

Since inflammation of the peritoneum usually starts from a pathological process in some organ of the abdomen, and becomes extended to the adjacent peritoneum, and thence to remote parts of the peritoneum, it is important to appreciate the local causes of peritonitis. These are found oftenest in the alimentary and genital tracts. Wounds of the stomach and intestines from without and from within, perforating ulcers and cancer, typhoid and dysenteric ulcers, perforating appendicitis, strangulated and invaginated intestine, are causes to be found in the gastro-intestinal canal. Puerperal, gonorrhœal, and menstrual infection may be extended to the peritoneum through the Fallopian tubes or through the lymph-vessels. The gonorrhœal lymphadenitis of an inguinal lymph-gland may serve as the starting point of a general peritonitis, as may the typhoid necrosis of an ileo-cæcal lymph-gland. Uterine and ovarian tumors are a frequent cause of a circumscribed peritonitis, and the latter especially, in consequence of a rupture or twist, may cause a general peritonitis. Inflammation, perforation, and rupture of the bladder and abscess of the kidney are causes proceeding from the urinary tract. Injuries, abscess, and echinococci of the liver, inflammation of the gall-bladder, with or without calculi, and inflammation of the pancreas are occasional causes of peritonitis. Infective necrosis, abscesses, and tumors of the spleen are also to be included among the local causes. A suppurative pleurisy or pericarditis may become extended to the peritoneum through continuous tissues, as may an erysipelas of the abdominal wall or an infective inflammation of the navel in the new-born child. A fœtal peritonitis sometimes occurs, attributed to syphilis, and Tolmatschew reports a case of peritonitis in a child three days old from a softened gumma of the bowel.

The local cause of a peritonitis in measles, small-pox, and intermittent fever may escape recognition, while an infectious necrosis of the spleen in typhoid or relapsing fever and a slough of the intestine in purpura may account for the peritonitis which sometimes occurs in these diseases.

A peritonitis may arise as a complication of nephritis, and its occurrence in scarlet fever is sometimes attributed to a concurrent nephritis, although it is becoming more probable that a concurrent acute nephritis and peritonitis may result from the same infectious cause.

Peritonitis in acute articular rheumatism may be the result of an infectious embolic necrosis in some structure covered with peritoneum, the latter being involved, or the peritonitis may be regarded as metastatic in the same sense that the arthritis or pericariditis is to be regarded as metastatic.

Apart from injuries and operations, the most frequent local causes of peritonitis are to be found in the gastro-intestinal tract, the female genitals, and the gall-bladder.

The following table is prepared to show in brief the conspicuous causes of peritonitis :

Traumatism.

Falls, blows, wounds, or operations, especially tapping, laparotomy, lithotrity, and litholapaxy, gynæcological and obstetrical explorations and operations.

Pathological Processes in the

Gastro-intestinal canal . . .	Gastritis, corrosive and phlegmonous.	
	Corrosive ulcer of the stomach and duodenum.	
	Typhoid, tubercular, and dysenteric ulcers.	
	Appendicitis.	
	Acute intestinal obstruction (strangulation, invagination).	
	Cancer of the stomach and intestines.	
	Perforation from foreign bodies.	
Biliary tract	Abscess	of the liver.
	Fibrous hepatitis	
	Cancer	
	Echinococcus	
	Inflammation,	calculous and cancerous, of the gall-bladder.
	Perforation,	

Pancreas	Inflammation.	
	Abscess.	
	Cyst.	
	Cancer.	
Spleen	Abscess.	
	Necrosis (typhoid, relapsing fever, and pyæmia).	
	Cancer.	
Genito-urinary apparatus	Inflammation of uterus, tubes, and ovaries (puerperal, gonorrhœal),	genital.
	Infectious menstrual fluid from the tubes,	
	Tumors of uterus and ovaries (twisted pedicle and ruptured cysts),	
	Gonorrhœal lymphadenitis,	
	Ectopic gestation,	
	Hæmatomata and hæmatosalpinx,	
	Cysts,	urinary.
	Cancer of bladder,	
	Cancer of kidney,	
Vascular apparatus	Aneurism of aorta.	
	Dilated and ruptured lymphatics and lacteals.	
Contiguous parts	Extension of suppurative pleurisy and pericarditis.	
	Tuberculosis of spine and pelvic bones.	
	Erysipelas of the abdominal parietes.	
	Gangrene of the navel.	

Infectious Diseases.

Measles, scarlatina, variola, intermittent fever, acute rheumatism, tuberculosis, hæmorrhagic purpura, scurvy, nephritis, congenital syphilis.

Morbid Anatomy.—In the early stages of acute peritonitis the subperitoneal blood-vessels are injected and minute hæmorrhages are seen. The peritoneum becomes opaque, its surface velvety and lustreless. Soft, yellow, fibrinous membranes adhere to the surface or unite opposed surfaces. These adhesions may resemble the threads of a spider's web or may form thick, curdy masses. A liquid exudation soon makes its appearance in the dependent portions of the peritoneal cavity, and may vary in quantity from a few ounces to several quarts. It is more thin, yellow, and transparent (serous), or thicker and more opaque (purulent), or red from the presence of red blood-corpuscles (hæmorrhagic).

The hæmorrhagic exudation occurs rather in tubercular or cancerous peritonitis or in cases of scurvy.

When the peritonitis is the result of the perforation of the digestive canal, the exudation is thin, opaque, greenish-gray, of an extremely offensive odor. A fæcal odor may also be present in the purulent exudation which has remained for some time in contact with the large intestine, although there be no perforation of the bowel. Gas also is present in the superjacent parts when the peritonitis is the result of a perforation of the gastro-intestinal canal.

At the outset of most cases of peritonitis the alterations are circumscribed in accordance with the local origin, and may remain so throughout. As the inflammation extends the peritonitis tends to become general. Even in general peritonitis the exudation gravitates toward the dependent parts, as the pelvis and loins, or lies between intestinal coils and remains fixed by the adhesions. In either general or circumscribed peritonitis the stomach and intestines become distended, sometimes enormously, with gas; their walls become œdematous and friable; the peritoneum is readily torn and is easily stripped from the muscular coat.

Symptomatology.—The symptom usually announcing the onset of an acute peritonitis is pain. This may be preceded by a chill, but is generally sudden and unexpected, especially in peritonitis from perforation, when it may be associated with a tearing sensation. It is apt to be intense, and is piercing, cutting, or griping, and becomes aggravated on motion of the patient, even from breathing, coughing, or vomiting. It is excited by peristalsis of the bowels, and is then often associated with the sound of moving air or liquid. The patient lies upon his back, often with the knees elevated, and dreads any physical examination through fear of an exacerbation of the pain, which may temporarily diminish. In rare cases the pain may be slight and cause no complaint. It is most severe in cases of peritonitis from perforation, and least intense in puerperal cases. It diminishes with the progress of the disease, especially with the appearance of the exudation.

It is frequently localized at the outset, and such localization is important in the usual secondary peritonitis, as directing attention to the seat and nature of the disease to which the peritonitis owes its origin. It is often referred to a point remote from its place of origin, usually to the vicinity of the navel, but its actual place of origin is often determined by the complaint of tenderness, often extreme, on palpation and on vaginal or rectal examination. Both pain and tenderness tend to spread with greater or less rapidity as the originally local peritonitis tends to become general, being most severe along the line of advance.

Vomiting usually takes place soon after the pain is felt, although it may be the first symptom. It is apt to persist throughout the disease. At first the contents of the stomach are expelled; then a bile-stained fluid is ejected; and finally the so-called fæcal vomit is likely to appear. The last is the homogeneous yellow, offensive contents of the small intestine, which enter the stomach in the later stages of the disease through an incompetent pylorus. The vomiting is readily excited by the entrance into the stomach of food or drink, even in small quantity, or by enemata. A sensation of nausea is likely to be constant, and is associated with more or less belching.

When the peritonitis affects the upper part of the abdomen, especially the surface of the diaphragm, hiccough may be obstinate, and, if it occur early, may prove most distressing as an aggravating cause of pain.

Abdominal distension is the next important early symptom of acute peritonitis, and may be present within a few hours after the incipient pain, immediately following which the abdominal muscles near the seat of the pain are firmly contracted, and the abdominal wall even retracted. The distension results from the gaseous formation and accumulation within the bowels, the result of a paresis or paralysis of the muscular coat of the intestines. It increases with the progress of the disease, and is the more extreme the thinner, weaker, and more flaccid the abdominal muscles. It is consequently likely to be greatest in puerperal peritonitis. On the contrary, it is less well marked in men with powerful muscles. The distension may become so great that the abdomen becomes dome-shaped, projecting above the thorax. The overlying skin is then smooth and shining, and the outlines of the distended coils may be seen. The diaphragm is forced upward, perhaps as high as the third rib. The apex of the heart is displaced upward and outward, and may be found in the fourth intercostal space, while the upper border of cardiac dulness may be found at the third rib. The lower lobes of the lungs also are pushed upward and backward, and may be prevented from expanding. The liver too is forced upward, and so rotated on its transverse axis that the upper border of hepatic dulness is not only as high as the third rib, but its area is considerably diminished, and may even be wholly lost. As the peritonitis progresses the gaseous distension may diminish, and be replaced to a certain extent by the fluid exudation, which first makes its appearance in the pelvis and flanks.

The mental condition of the patient at the outset is one of suffering and anxiety. The intelligence often remains unaffected till death, which often takes place without warning or may be preceded by mild delirium or by stupor.

In peritonitis from perforation into the normal peritoneal cavity symptoms of collapse quickly follow the initial pain. The eyes become sunken, the nose pinched, the face and extremities cold, the voice husky, and the pulse rapid and feeble, perhaps not to be counted. These symptoms may also occur, although later and more gradually, in peritonitis without perforation or when, the rapid progress of the peritonitis is limited by adhesions.

The pulse early becomes rapid, small, and weak. While the peritonitis is circumscribed it may remain near 100 beats in the minute, but as the peritonitis becomes general it rises to 130 or 140, and may become imperceptible.

The respiration is rapid and superficial, 30 to 40 a minute. It is made difficult by the ascent of the diaphragm, the compression of the lower lobes of the lungs, and the enfeebled action of the heart. It is soon painful from the sensitiveness of the nerves of the inflamed peritoneum covering the diaphragm and the anterior wall of the abdomen.

The temperature has no constant range. It is usually to be found between 100° and 103° F., but may be lower in serious cases, even subnormal in collapse, and in rare instances shortly before death a temperature of 110° F. or more may occur. There may be slight daily variations, or the evening temperature may be two or three degrees higher than that of the morning.

The tongue at the outset is moist and covered with a thin white coat. Later it becomes dry and the surface brown and cracked.

There is no appetite, and food is often quickly regurgitated, either in small quantity or after a lapse of hours in large amounts and apparently undigested.

The bowels are usually constipated from paralysis of the muscular coat, although the paralysis, as suggested by Bauer, is not universal, since the presence of fecaloid vomiting indicates the existence of peristalsis in the small intestine. Loose dejections may be present in the early stages of peritonitis from peritoneal irritation, and diarrhœa may occur in the later stages from associated enteritis. In puerperal cases frequent loose dejections are the rule.

Micturition is likely to be frequent and painful, with a scanty flow of urine when the peritoneal coat of the bladder is involved in the inflammation. Later, retention, requiring the use of a catheter, may occur from paresis of the muscular coat of the bladder and from the use of opium. The quantity of urine is diminished. This secretion is dark-colored, strongly acid, and of high specific gravity. A trace of albumin is frequent. Indican is present in large quantity, and may be shown by Jaffé's method : Equal parts of urine and hydrochloric acid are to be shaken with two or three drops of fresh concentrated solution of chloride of lime. The mixture becomes of a dark-blue, even bluish-black, color.

On palpation of the abdomen at the beginning of peritonitis the walls are tense and resistant; as the abdomen becomes distended the tension persists, but the resistance diminishes. With the formation of a fibrinous exudation a friction is sometimes to be felt. More frequently a fluid exudation is to be determined by the transmission of a wave of fluctuation, especially at the dependent portions of the abdomen. That fluctuation may exist, it is necessary that a considerable quantity of fluid should be present and the abdominal tension slight. Fluctuation may be simulated in the absence of any considerable quantity of fluid in the peritoneal cavity if the intestines are distended with liquid or if there be abundant fat-tissue or fluid in the abdominal wall.

Percussion of the distended abdomen gives a clear tympanitic sound in those parts where the distension is due to gas in the intestines. Considerable importance has been attached to the replacing of the area of hepatic dulness by a tympanitic sound as evidence of a perforation of the stomach or intestine. This sign is of but little weight, since it may be produced by the interposition of intestine between the liver and the abdominal wall, and by the displacement and rotation upward and backward of the anterior border of the liver in extreme gaseous distension. If the liver be attached to the abdominal wall by old adhesions, gas cannot enter between the diaphragm and liver. It is also important in the appreciation of the value of this disappearance of liver dulness to know the position of the intestines in the patient during health. As the peritoneal exudation becomes fluid or the intestines become distended with liquid contents, the percussion-note becomes dull or flat, usually in the dependent portions of the abdomen. If the dull or flat sound be due to free fluid, the line of dulness changes with the position of the body. If the fluid be encapsulated by adhesions, the dull region remains fixed. Absent dulness does not indicate failing exudation, since the latter may be separated from the abdominal wall by overlying intestine with gaseous contents.

On auscultation of the abdomen a loud gurgling is to be heard as the contents of the intestines are moved. A friction sound may also be heard at times, from the rubbing against each other of the inflamed peritoneal surfaces. This rubbing may be synchronous with the respiratory movements or may result from intestinal peristalsis.

The **course** of acute general peritonitis is usually rapid and unfavorable. Death generally takes place during the first week or in the early half of the second week. If the patient survive to the end of this time, the disease tends to become chronic. Chronic peritonitis may result fatally in the course of weeks or months by a progressing wasting and exhaustion. The results of chronic peritonitis may remain indefinitely as thickened fibrous patches and plates, with associated atrophy of the spleen or liver. Stricture of the intestine with symptoms of chronic obstruction may result from the thickened peritoneum. Bands and cords may remain as permanent fibrous adhesions, which may impair the movement of the intestines or produce acute strangulation. Such adhesions between the uterus, tubes, and ovaries may cause sterility or ectopic gestation, and interfere with intra-uterine gestation and childbirth. These adhesions, and especially those around the appendix and gall-bladder, may favor recurrent attacks of localized peritonitis.

Diagnosis.—As a rule, the diagnosis of peritonitis is easily made. The presence of abdominal pain, vomiting, tympany, and fever gives a grouping of symptoms which need merely the evidence of the exudation to be regarded as characteristic. Difficulties are likely to arise only in the earliest stage, when sudden and unexpected pain and vomiting are the only striking symptoms. These may be due to the various forms of colic, gastric, intestinal, renal, biliary, and uterine. Gastro-intestinal colic frequently has a well-defined cause, and is often relieved by pressure. Renal colic is limited to the urinary tract, and is likely to be associated with gravel, calculi, or blood in the urine. Biliary colic is seated near the gall-bladder, which is often to be felt. Jaundice may accompany the pain or gall-stones may be found in the fæces. Uterine colic is associated with menstruation, pregnancy, or a tumor, and the pain is more intermittent than in peritonitis. The pain of acute intestinal obstruction may closely simulate that of acute peritonitis. The former is to be excluded rather by the presence of a probable cause of peritonitis. In obstruction of the large intestine there is no excess of indican in the urine, as occurs in acute diffuse peritonitis.

In rare cases pain may be absent, and the diagnosis will then more especially depend upon the presence of the exudation. It is to be remembered that a pregnant uterus, an ovarian tumor, an echinococcus cyst, and a full bladder have each been mistaken for a peritonitic exudation.

For therapeutic purposes it is most important to diagnosticate the cause of the peritonitis, and to decide whether the inflammation be circumscribed or spreading. The cause of a peritonitis is to be arrived at by a knowledge of the history of the individual case, a determination of the seat of original tenderness, and a knowledge of the etiology of peritonitis.

The spreading of the peritonitis is indicated by the extension of the pain and tenderness, the advance of the exudation, and is often to be determined only by vaginal or rectal examination. Additional evidence is furnished by the persistence or increase of the vomiting, tympany, and symptoms of collapse.

Prognosis.—Acute general peritonitis is always extremely grave, and its result depends essentially upon the cause of the inflammation, the quality of the exudation, and the associated disease.

Peritonitis from perforation of the stomach and free portion of the intestine into the normal peritoneal cavity is almost invariably fatal. Puerperal peritonitis and that following instrumental abortion are not infrequently fatal, especially when the exudation is abundant and rather purulent than serous or fibrinous. Circumscribed varieties of peritonitis always offer a more favorable prognosis than the diffuse forms, and may recover under medical treatment, while a spreading peritonitis may demand the most radical surgical measures. The cessation of vomiting and the return of defecation are favorable signs in the prognosis of the individual case.

Treatment.—The first indication in the treatment of peritonitis is to relieve pain. The measures employed for this purpose are often of service in circumscribing the inflammation, thus rendering the prognosis more favorable. These measures consist in opium internally and heat or cold externally. The dose of opium taken into the mouth of an adult should be 1 grain in pill or 15 drops of laudanum, or the same quantity of the deodorized tincture of opium. It should be repeated every hour until the pain is relieved or toxic symptoms are manifested. These symptoms are contracted pupils, slow respiration, and stupor. It is always to be remembered that the opium is not given to cure the peritonitis, but to relieve the pain; that the pain usually becomes lessened without drugs as the exudation becomes more abundant; and that the opium is absorbed but slowly in peritonitis. It is customary to say that a patient with peritonitis tolerates more opium than one not suffering from this disease. This tolerance, however, only applies to opium or its preparations taken by the mouth. Subcutaneous injections of morphine act with great rapidity, and their effect prevails for several hours after their administration. It is therefore unwise to continue the frequent use of opium by the stomach in increasing doses until stupor appears, as a large quantity of unabsorbed opium may then be present in the stomach or intestine. When the pain is relieved the dose of opium should be continued only at such intervals as will ensure freedom from pain. If in consequence of persistent vomiting the opiate be not retained in the stomach, subcutaneous injection of one-fourth-grain doses of morphine, or opiated or morphinated rectal suppositories, should be used.

Heat is usually preferred to cold as a local application by the patient. It may be applied as flaxseed poultices, not too large and not so thick as to be burdensome. In case of associated meteorism a teaspoonful or two of equal parts of sweet oil and oil of turpentine may be smeared over the inner surface of the poultice before it is placed on the abdomen. If the poultice be uncomfortable on account of its weight, turpentine stupes may be used in its place. Both poultices and stupes are to be covered with oiled silk or some other non-conductor of heat and moisture: a folded newspaper even may thus serve a useful purpose.

Heat may also be applied in a rubber bag filled with hot water. The latter method is useful chiefly in cases of circumscribed peritonitis in the flanks or subthoracic regions, where the weight of the bag and its contents are not inconvenient. Care should be taken that the heat, however applied, should not be so extreme as to burn the skin.

Cold will sometimes prove more agreeable than heat. It is most conveniently applied to the abdomen as ice broken into small bits and placed in a rubber bag, or mixed with linseed meal, forming an ice-poultice. In the latter case, or where cracked ice is wrapped in a folded cloth, care should be taken to collect the water resulting from the melting of the ice in some suitable absorbent, as a woollen or cotton cloth.

Vomiting may be relieved by the subcutaneous injection of morphine, aided by pellets of ice taken into the stomach, or by teaspoonful doses of hot water or coffee, or by denying all food and drinks. Even if no considerable quantity of liquid be swallowed, vomiting may persist, the intestinal contents being forced into the stomach through an insufficient pylorus by the pressure of the tympanitic intestines or by a reversed current produced by the contraction of the wall of the upper portion of the bowel, forcing the intestinal contents against a lower segment obstructed by paralysis or compression. Kussmaul claims great relief in such cases by siphonage of the contents of the stomach through an elastic tube.

Hiccough may also be relieved by subcutaneous injections of morphine and by the internal administration of ice. If it prove unusually distressing and enfeebling, occasional whiffs of ether or chloroform may diminish its intensity. Five to ten grains of phenacetin or of antifebrin have proved efficient in controlling this symptom.

Meteorism may prove one of the most obstinate symptoms, especially when unrelieved by external applications of turpentine. Lavage of the stomach may relieve the distension of the upper portion of the abdomen, and rectal injections of tepid or cold water may empty a distended colon. The intermediate portion of the bowel will be relieved but little by either of these methods. The passage of a soft catheter or tube into the rectum will frequently favor the expulsion of a certain quantity of gas. In extreme cases, however, recourse must be had to frequent punctures of the distended bowel with a small hollow needle. The danger of extravasation or the escape of gas into the peritoneal cavity when this measure is employed is comparatively slight.

Constipation which is unrelieved by rectal enemata should be left untreated, except early in those cases of peritonitis of puerperal or operative origin where intestinal causes may be excluded. The use of laxatives or cathartics in peritonitis of intestinal origin where perforation or mechanical obstruction exists has frequently been immediately followed by a rapidly fatal result.

The diet in peritonitis should be of the blandest sort. The patient often rebels at the thought of food, since his experience soon teaches him that it is not likely to be retained. It should consist mainly of milk, either hot or cold, peptonized or diluted with lime-water. Aërated saline waters, as Seltzer, Apollinaris, Vichy, or carbonic-acid water, may be used instead of lime-water, although they are often objected to from the presence of the gas. Some patients prefer them as fresh as possible, while others object to swallowing them unless the gas has first been allowed to escape.

Alcoholic stimulants are demanded in the later stages of peritonitis, and may be given as spirit, dry champagne, or sherry, diluted with water or milk as the patient may prefer.

Of late years attention has been drawn to the use of saline laxatives in acute peritonitis, principally in consequence of the experience of Tait in the treatment of peritonitis following surgical operations. This surgeon thinks opium harmful in such cases, and advocates the early use of saline cathartics in hot water, rather to avert than to cure peritonitis. Seltzer (*Phila. Med. News*, 1891, lviii. 26) states that this treatment was recommended by Abernethy in 1830 when threatening peritonitic symptoms followed the operation for adherent omental hernia.

The use of salines has also been advocated for many years in the treatment of puerperal peritonitis, having been introduced by Seyfurt. It was based upon the observation that in these cases the onset of a profuse diarrhœa was followed by a diminution of pain, tympany, and fever. Even in the operative cases, however, it is recognized that the saline treatment, to be of value, must be employed early, before there is much abdominal distension or excessive vomiting, while in severe cases of acute puerperal septicæmia diarrhœa is caused with difficulty, and, if produced, is of but little benefit. In suitable cases of beginning peritonitis of non-intestinal origin the published evidence warrants a trial of the saline treatment. The administration of a teaspoonful of Epsom salts or a Seidlitz powder every two hours until the bowels are moved may be tried. If no relief follow, the treatment with opium should be resorted to.

The measures above recommended relate simply to the medical treatment of acute peritonitis, and are intended to apply only to the early stages of the disease, when there is hope that the peritonitis may become circumscribed.

In all cases of spreading peritonitis with urgent symptoms, whatever may be the cause—which is often not to be ascertained with any degree of certainty—

surgical treatment is that which offers the most hope. The efforts at circumscribing the peritonitis are frequently to be regarded as leading to an eventual surgical operation, which offers a more favorable outlook the more the inflammation is limited to the part of the peritoneal cavity exposed by the surgeon's knife.

CHRONIC PERITONITIS.

Like acute inflammation of the peritoneum, the chronic variety may be both circumscribed and diffuse. Either of the latter varieties may be the result of the acute form or they may be latent from the outset. In the former instance it is manifested rather by a protracted convalescence from acute peritonitis than by any marked characteristic signs. The symptoms of the acute stage gradually diminish in severity until more or less complete recovery takes place. This recovery is more complete in the case of the circumscribed than in that of the diffuse variety. Even in the former instance the absence of complete recovery may be indicated by localized pain associated with disturbance of function of the organ whose peritoneal covering is conspicuously affected. Chronic constipation, acute intestinal obstruction, disturbances of menstruation, malpositions of the uterus, distention of the gall-bladder, with occasional attacks of jaundice, may all give evidence of the results of a previous attack of acute peritonitis.

A latent attack of chronic circumscribed peritonitis is rather discovered after the death of the individual, when fibrous adhesions, patches, and plates may be found between and upon the surface of the abdominal viscera and the neighboring peritoneum. Thus the liver may be united to the diaphragm, the gall-bladder to the omentum, the spleen to the diaphragm and abdominal wall. The omentum may be found attached to the pelvic viscera or to the peritoneum of the pelvic wall. The uterus, ovaries, and tubes may be united together and to the broad ligament. Loops of intestine may be adherent to each other or to the peritoneum covering the pelvic organs and abdominal wall. The vermiform appendix may be buried in fibrous tissue or be displaced above, below, to one side, and be firmly attached to the adjacent peritoneum.

In chronic diffuse peritonitis directly continued from the acute attack the absorption of the exudation is protracted: not only are fibrous adhesions, often of extreme density, present in various parts of the abdominal cavity, but semi-fluid or partly inspissated, even calcified, exudations may also be found encapsulated between opposed peritoneal surfaces. Such exudations may escape through perforations into the intestine, bladder, or abdominal wall, or may serve as a centre from which a recurrent attack of acute peritonitis proceeds.

Of late years especial attention has been directed to the recognition of cases of chronic diffuse peritonitis with profuse liquid exudation which have hitherto been confounded with ascites or with tuberculous, cancerous, or sarcomatous peritonitis. Galvagni, Rehn, Bauer, Henoch, Vierordt, and Leyden have published more or less extensive communications on the subject, and substantially agree in their observations.

Etiology.—The causation of this affection is exceedingly obscure. Trauma, exposure to cold, profuse and protracted diarrhœa, measles, typhoid fever, syphilis, infection from a vulvo-vaginal catarrh, have been mentioned as exciting or predisposing causes, but a more intimate relation than that of sequence has hardly been established.

This variety of chronic, idiopathic, or essential peritonitis usually attacks girls after the age of three years, and has been frequently noticed to occur at the beginning of puberty. It is not unlikely, as suggested by Henoch, that many of the cases of supposed peritoneal tuberculosis in the adult, especially those with abundant liquid in the peritoneal cavity, may belong in this category.

Symptoms.—The patient may appear in excellent health, showing no loss of flesh, strength, or color even in the presence of extreme abdominal enlargement, which is the first constant, characteristic symptom. In other instances there may be loss of appetite, pallor, and progressive emaciation. There is little or no fever, pain, or tenderness.

The abdominal enlargement increases often to an extreme degree, usually slowly, in the course of months, sometimes in the course of a few weeks. There may be occasional variations in the degree of enlargement in the same case, the abdomen being now smaller and again larger, without any corresponding change in the general symptoms of the disease.

The physical examination of the abdomen indicates the presence of free fluid in the abdominal cavity. Such fluid is either serous, sero-fibrinous, or sero-purulent. It is richly albuminous, thus resembling rather an exudation than an effusion. In the course of months the fluid may become absorbed in connection with continued diarrhœa or marked diuresis. Palpation of the abdomen may then reveal dense, rounded masses in the abdomen, callous thickenings of the peritoneum covering the intestines, omentum, or mesentery.

Morbid Anatomy.—In a case reported by Hirschberg, which proved fatal from intercurrent disease, the peritoneum was found to be unaltered except over a portion of the colon, where it formed an irregular thickening without associated constriction of the bowels. In another lately reported by Henoch laparotomy was performed, and the peritoneum was found studded with small nodules presenting the gross characteristics of tubercle. The microscopic examination showed neither giant-cells nor tubercle-bacilli, merely fibrous and granulation tissue.

Diagnosis.—Since the essential characteristic of this affection is distention of the abdominal cavity with fluid, it is likely to be confounded with ascites and peritoneal tuberculosis. The former is excluded by the absence of jaundice, clay-colored stools, continued gastro-enteric disturbances, itching skin, and hæmorrhages.

Peritoneal tuberculosis may be more closely simulated, and at times to be ruled out with difficulty. There is no evidence of tuberculosis elsewhere in the patient or the family. The emaciation is less constant, fever, tenderness, and pain are often absent, and the progress of the abdominal swelling is unattended with the frequent signs of progressing weakness. The advance of peritoneal tuberculosis is more rapid than that of this variety of chronic peritonitis.

Prognosis.—The prognosis is favorable, though months may elapse before the abdominal swelling disappears.

Treatment.—The medical treatment consists essentially in nourishing, easily digested food; tonics, as iron, quinine, arsenic, saline laxatives in case of constipation, and diuretics. But little relief is likely to result from local measures designed to remove the fluid, as blisters or iodine. Vierordt claims that benefit was derived from frictions of the abdomen with gray ointment, which produced a marked diuretic action.

Puncture of the abdominal wall is the most rational form of local treatment, and has proven successful in certain instances. The fluid may reaccumulate and repeated punctures become necessary. In a case reported by Henoch more than double the quantity was drawn at the third puncture than at the first. The abdomen rapidly refilled, laparotomy was performed, and recovery soon followed.

Brief allusion may be made to two rare varieties of chronic peritonitis of more anatomical than clinical interest. The first is that described by Friedreich as chronic hæmorrhagic peritonitis. It followed repeated punctures for ascites. The visceral and parietal peritoneum was covered with a thick, granulated, adherent membrane; numerous nodules of extravasated blood were found between the layers of false membrane.

Werth applies the term *pseudo-myxoma peritonei* to the presence of the gelatinous contents of a ruptured ovarian cystoma in the peritoneal cavity. In consequence of the irritation excited by this material, fibrous adhesions are formed between the peritoneal surfaces. These adhesions, fibrous and vascular, traverse the gelatinous substance, holding it enmeshed. Delicate fibrous membranes may also surround bits of gelatinous material, which thus become attached to the surface of the intestine as polypoid appendages.

TUBERCULAR PERITONITIS.

A distinction is sometimes drawn between tuberculosis of the peritoneum and tubercular peritonitis. The former term was applied to the presence of miliary tubercles in the peritoneum, the latter to the coexistence of tubercles and such inflammatory products as serum, fibrin, or fibrous tissue. The distinction is arbitrary and unnecessary. Miliary tubercles are now regarded as inflammatory products due to Koch's bacilli within tissues, and there is no definite dividing-line between tubercles with no exudation and tubercles with abundant exudation.

Etiology.—Tubercular peritonitis is to be regarded as almost invariably secondary to tuberculosis elsewhere in the body. It may occur as a part of a genital tuberculosis, especially in the female, but it is of chiefest importance when its symptoms and lesions predominate over those of tuberculosis elsewhere in the body. It is then to be practically regarded as local, although usually extended from elsewhere.

Pribram states that of 165 cases of peritoneal tuberculosis examined after death, 87 were attributed to intestinal tuberculosis, 8 to tubal and uterine tuberculosis, 5 to old and recent bone-tuberculosis, and the rest to pulmonary and glandular tuberculosis. König states that in 107 autopsies of tubercular cases the lungs were simultaneously diseased in 99 cases, the pleura in 60, the intestines in 80, the ileo-peritoneal glands in 44, the spleen in 40, the kidneys in 38, the liver and suprarenal capsules, each, in 6 cases. The extension of the disease to the peritoneal cavity thus usually takes place from the intestines, lungs (through mediation of the pleura), retro-peritoneal lymph-glands, and the Fallopian tubes. Although in the 107 cases 7 per cent. were females, in 131 cases of laparotomies in tubercular peritonitis 92 per cent. were females. According to Osler, this affection is most common between the ages of twenty and forty, and it is rare in old age. Many writers refer to the frequent concurrence of chronic fibrous hepatitis and tubercular peritonitis, the former thus being regarded as a predisposing cause.

Morbid Anatomy.—The tubercles are the characteristic feature of tubercular peritonitis. The associated changes vary largely in accordance with the quantity and quality of the exudation and with the extent and duration of the process.

When tubercular peritonitis is part of an acute miliary tuberculosis, the peritoneum in general will be studded with gray, glistening, translucent granules, smaller than a pin-head and projecting slightly above the surface. The peritoneum otherwise may be comparatively unaltered—at the most, perhaps, injected. There may be a few ounces of clear yellow fluid in the peritoneal cavity. Similar appearances may be the result of other miliary granules than tubercles, especially small fibromata. In all doubtful cases it is, therefore, necessary that the granules should be examined histologically. Bacilli should also be sought for, and in important cases the negative result of such a search should be supplemented by inoculation-experiments. This is all the more necessary since there are fibrous tubercles not to be distinguished by gross appearances, and even by microscopical examination, from the fibrous nodules occurring in chronic peritonitis. The more the tubercular peritonitis tends to be independent of a general tuberculosis, and the more it represents the chief tubercular changes in the body, the more conspicuous are the alterations of the peritoneum and the more abundant and varied is the character of the exudation.

The peritoneum is not only studded with tubercles which tend to become clustered into patches and nodules, but it also becomes thickened, injected, ecchymosed, and often covered with a layer of fibrin. The agglomerated tubercles are opaque, either grayish-white or yellow, cheesy. The omentum may be shrivelled into a dense sausage-shaped tumor crossing the abdomen above the navel. The mesentery, in like manner, may be thickened and contracted, and the attached small intestine, especially when united by adhesions, may form a globular mass attached to the spine.

The exudation is either serous or fibrinous, rarely purulent, though not infrequently hæmorrhagic. Its quantity is greatest in the absence of adhesions, which, however, are constantly present in the more protracted cases. Fibrinous adhesions are a part of the sero-fibrinous exudation, and not infrequently conceal the underlying tubercles. They may even encapsulate the liquid exudation, several collections of which may thus be separated from each other. The fibrous adhesions serve to unite firmly the peritoneal surfaces to each other, and tubercles may be found within them. They may be so voluminous and dense as to be mistaken for cancerous or sarcomatous growths when the abdomen is palpated. They may be felt as indurated masses or as bands and cords. In their presence, and with little or no liquid exudation, the abdominal wall may be retracted and the peritoneal cavity more or less extensively obliterated. With the circumscribing of the liquid exudation the outer surface of the intestine may become perforated, and an interchange of contents take place between the sac and the intestine. Intestinal fistulæ thus arising may be continued through the abdominal wall, and the vermiform appendix may be found at the bottom of such an opening, its tip being free and movable.

Circumscribed tubercular peritonitis not infrequently exists, limited to the peritoneal surface of tubercular ulcers of the intestine, or to the pelvic peritoneum in the vicinity of tubercular Fallopian tubes, or to the under surface of the diaphragm in connection with tubercular pleurisy or pericarditis. Localized tumors may thus be formed, especially in the pelvis and right iliac fossa.

Symptomatology.—The chief symptoms of tubercular peritonitis are abdominal pain and tenderness, general abdominal enlargement, or a circumscribed tumor, and fever. The onset of these symptoms may be sudden and somewhat violent, or so gradual that the enlarged abdomen first attracts attention, and the tubercular nature of the process may first be discovered when the abdomen is opened for the purpose of removing a supposed ovarian tumor. Periods of intermission, with recurrence of the symptoms, are noticeable in certain cases.

The abdominal pain is usually slight, and is then associated with little or no tenderness. It may be wholly absent, or, again, may be severe at the outset and accompanied by marked tenderness. When present it may be circumscribed or diffuse.

The fever, as a rule, is slight. The temperature may be normal, even subnormal for a long time. When exacerbations and remissions of pain and swelling occur, the temperature will show corresponding variations.

The abdominal enlargement, when present, is due to the liquid exudation or to meteorism, or, more frequently, to both. Its degree may therefore vary considerably in individual cases, and from time to time in the same case. The quantity of liquid may be very large, the intestines floating in it, and it may remain free, with distinct fluctuation, throughout the disease. The liquid exudation tends to become eventually encapsulated by adhesions, even if such are lacking at the outset. Large or small collections of fluid, few or many, may thus be circumscribed, suggesting single or multiple cystic tumors. In connection with gaseous distension of the bowels projecting tumors may result, which may lie above or below, on one side or on the other, and which remain fixed when the position of the patient is changed. Such tumors are rounded and elastic, and are not to be confounded with the resistant thickenings of the abdominal wall or the sausage-shaped contractions of the omentum. Rectal or vaginal examination will not infrequently reveal an indurated condition of the pelvic peritoneum corresponding to that to be felt on abdominal palpation.

The more abundant and dense the adhesions, the greater the probability of digestive disturbances, manifested by loss of appetite, nausea, diarrhœa, or constipation. Symptoms of intestinal obstruction may occur, and have led to a laparotomy for their relief.

The more latent and gradual the onset of tubercular peritonitis, the more prolonged the course, which may extend over a long period of months. There may be progressive wasting and debility, markedly increased in those cases where intestinal fistulæ are established. Death may result from the presence of tubercle in other organs or from the accession of a perforating peritonitis to the tubercular peritonitis.

Diagnosis.—The difficulty of diagnosticating tubercular peritonitis is apparent when it is remembered that in the majority[2] of the cases where this affection has been found at a laparotomy the diagnosis of some other affection had been made. Especially has it been mistaken for an ovarian cystoma or for some abdominal tumor with liquid contents. All the symptoms may occur in chronic peritonitis of non-tubercular origin, and the latent course and abundant liquid exudation have frequently suggested the presence of ascites.

Most important points in making a diagnosis are a consideration of the etiology, and the exclusion of causes of ascites and abdominal tumors. Etiology is more important than exclusion. Most cases of subacute and chronic general peritonitis of non-traumatic origin not dependent upon antecedent acute peritonitis, and not attributable to cancer or sarcoma, may be tubercular. They probably are tubercular if there be antecedent or concurrent pulmonary, intestinal, genital or genito-urinary, glandular, or osseous tuberculosis. The associated presence of pleuritic effusion favors the view of the tubercular nature of the abdominal process. Most important in the diagnosis is the exclusion of the causes of ascites and of ovarian cystoma, abdominal cancer, or sarcoma.

Ascites is excluded by the frequent presence of a certain degree of abdominal pain and tenderness, more or less protracted fever, more emaciation, frequent indurations, and by the absence of jaundice, gastro-intestinal hæmorrhages, and splenic enlargement. Ovarian cystomata are to be eliminated by the rapidity of enlargement and by the more rapid and more profound digestive disturbances and loss of flesh and strength. Malignant disease of the abdomen progresses even more rapidly than tubercular peritonitis, and is likely to be secondary to malignant disease of some one of the abdominal organs, whose functions may be conspicuously impaired.

Prognosis.—Although a fatal result is to be anticipated as the usual outcome of this disease, the occasional temporary improvement under treatment, and at times the probability of a spontaneous improvement, make the prognosis less definitely grave. Cases are recorded where the exudation in supposed tubercular peritonitis has disappeared, in one instance for a period of three years. In another case two years after such disappearance the patient died of general tuberculosis, while in a case mentioned by Pribram a year and a half after the disappearance of the exudation laparotomy was performed for the removal of a parovarian cyst, and the remains of the tubercular process were found. The latter proved to be tubercular by microscopic examination and by the discovery of the bacilli.

The results of laparotomy of late years have apparently made a favorable prognosis the rule. Cures have been claimed in 70 to 80 per cent. of the cases operated upon. Within the past three years, however, doubt has again become necessary with reference to the curability of tubercular peritonitis. Spaeth, Löhlein, Henoch, and Vierordt have called attention to the faulty nature of the evidence on which the reported cures were based. That such a claim should be definitely established it is necessary that the granules present should contain the structure of tubercle and Koch's bacilli, or produce tubercle when inoculated. Such absolute evidence has only been rendered possible since Koch's discovery concerning the causation of tuberculosis. Of the numerous cases published since 1882, but few have shown the evidence necessary to permit them to be accepted as cases of unquestionable tubercular peritonitis.

Treatment.—The treatment is essentially constitutional and symptomatic. Rest, nourishing and easily digested food are essential.

Pain is relieved by opiates and fomentations, constipation by laxatives, diarrhœa by opiates. The last symptom may prove of benefit in diminishing the abdominal swelling by the absorption of fluid and the expulsion of gas.

Clark recommends the application of iodine and olive oil, 7 to 30 grains of the former to an ounce of the latter, to be applied three times daily. Vierordt favors inunction with mercurial ointment. Pribram prefers gentle friction, once daily, with green soap and a little water: the navel and hairy parts of the abdomen are to be avoided: the skin is to be covered with oiled silk or thin rubber cloth after the applications, which are to be continued until the skin becomes hard and scaly. Both he and Vierordt claim that under such local treatment the liquid exudation may be absorbed, the solid masses disappear, the fever subside, and the appetite improve. Such improvement is likely to be but temporary.

Puncture of the abdomen has proved efficient when there was abundant liquid exudation, and may need to be repeated several times in consequence of the return of the fluid.

Laparotomy is the most favored method of attempts at radical treatment, and, even if it accomplishes no cure, may produce rapid relief, extending over a period of years. It permits the more complete evacuation of the fluid, and may lead to the removal of a local tubercular process—*e. g.* the tubercular tubes. If the chronic peritonitis be simple and not tubercular, the prospects of speedy relief and ultimate cure from laparotomy are so great as to demand the operation. It is exploratory in the first instance, and may prove a cure of simple peritonitis, and a palliative, to say the least, in tubercular peritonitis.

TUMORS OF THE PERITONEUM.

Tumors of the peritoneum may be divided into those which proceed from the free surface and those which grow from between the folds of the omentum and mesentery. The former include those which are malignant, producing a cachexia—cancer, endothelioma, sarcoma, malignant adenoma (papillary cystoma)—as well as those which are benignant, producing merely mechanical disturbances—cystic, dermoid, and teratoid tumors. The omental and mesenteric tumors are the myxoma, lipoma, fibroma, sarcoma, hæmangioma, chylangioma, and cysts, entero cysts and serous-cysts.

Those tumors which are deserving of especial consideration in a work on the practice of medicine are the malignant peritoneal growths, which, for clinical convenience, may be described under the term of peritoneal cancer.

CANCER OF THE PERITONEUM.

Cancer of the peritoneum is usually a disease occurring after middle life, and is sometimes a primary growth, though usually secondary to cancer in organs covered by peritoneum. The primary tumor is usually in the stomach or intestine, especially in the cæcum and large intestine. It may proceed from the ovaries or uterus, from the kidney, pancreas or gall-bladder, or suprarenal capsule. It may also originate in the œsophagus or be continued from the retroperitoneal glands. The cancerous growth is usually continued directly into the peritoneal cavity, or it makes its appearance in the form of metastatic nodules.

The varieties to be found are the hard, fibrous ; the soft, medullary ; the pigmented, melanotic ; and the colloid, hyaline. The last mentioned is the most often found. The growth is rapidly extended from the starting-point by continuous and discontinuous growth until numbers of patches, nodules, and shapeless masses are to be found. Numerous minute granules of miliary cancer are sometimes to be seen, closely resembling miliary tubercles. The omentum and mesentery are favorite seats of the cancerous growth, the former sometimes being transformed into a bulky mass of tissue weighing many pounds. The peritoneum near the navel and in Douglas's fossa is likely to be the seat of the growth, the latter region being affected even before extensive disease has made its appearance elsewhere in the peritoneum.

Peritoneal cancer is usually associated with evidences of an inflammatory exudation or with a dropsical effusion. In cancerous peritonitis the liquid exudation is frequently hæmorrhagic, while in cancerous ascites a milky fluid may be present from fatty degeneration of the cells of the cancer. Fibrous bands and threads and peritoneal thickening are frequently associated with the cancerous growth, and the peritoneal cavity may become almost entirely obliterated by the numerous adhesions.

Symptoms.—Cancer of the peritoneum produces no characteristic symptoms. It may pursue its course painlessly, without the formation of tumors, and first be recognized after death. The symptoms connected with it are rather those of the associated peritonitis and ascites, and are chiefly of a mechanical sort, the peritoneal thickening and adhesions producing constipation and colic. The obstructed movement of the bowels may result in meteorism. An abundant liquid exudation, whether ascitic or peritonitic, causes the disturbance of respiration and circulation mentioned in the consideration of these subjects. Fever is present in cancerous peritonitis, but its course and range are so similar to that of tubercular peritonitis as not to be discriminated. The longer the disease continues the more likely to arise are the symptoms of cachexia, the wasting, pallor, and weakness.

Symptoms of severe hæmorrhage may result from the rupture of the vessels in the cancerous masses, or symptoms of a rapidly progressing peritonitis may result from perforation of the bowel underlying the tumor.

Palpation often makes apparent induration or tumors in various parts of the abdomen, either resistant or floating in fluid. The quantity of fluid may be so great that the tumors are first recognized after its withdrawal. In this fluid large and irregularly shaped cells, usually with fatty or hyaline contents, may be found. Gelatinous material may sometimes be aspirated, in which a cancerous or sarcomatous tissue is to be recognized. A rectal or vaginal examination is especially important in the early stage of peritoneal cancer, from the frequency with which the pelvic peritoneum is then diseased.

The **diagnosis** is based upon the recognition of tumors, usually movable and associated with symptoms of preceding disturbance of function in some one of the organs from which the disease has proceeded. It is confirmed by the recognition of cancerous tissue in fragments, which may be removed through a trocar. When associated with ascites the tumors may be first recognized after removal of the fluid. If there be an abundant fluid exudation without tumor, a differential diagnosis between peritoneal cancer and tubercular peritonitis may be impossible. In favor of cancer would be a more rapid progress with a greater degree of cachexia, while in favor of tubercular peritonitis would be a family and personal history of tuberculosis, with evidence of this disease in the favored parts of the body, as the lymph-glands, the lungs, intestines, genitals, and bones.

Prognosis.—The outcome is fatal, usually in the course of weeks or months, since it commonly represents the terminal stage of visceral cancer. The progress is much slower, though equally fatal, when it occurs as a primary affection. A rapidly fatal result is to be expected when perforation or intraperitoneal hæmorrhages occur as complications.

Treatment.—There is but one consideration in treatment—viz. the patient's comfort. This is to be obtained by opium for the relief of pain, mild laxatives to control constipation, and a non-irritating diet. The more closely the symptoms simulate those of peritonitis, acute or chronic, the more nearly should the treatment prescribed for these affections be followed. If the peritoneal fluid be sufficiently abundant to produce discomfort, it may be removed by tapping, especial care being taken to avoid puncturing an adherent coil of intestine. The fluid will soon return, and the more frequent the tappings the more rapid the patient's exhaustion. Not infrequently the cancerous growth will extend into the abdominal wall along the track of the trocar.

VERMINOUS PARASITES.

Of the verminous parasites which may be found in the peritoneal cavity, the echinococcus alone is of practical importance. The cysticercus cellulosæ and pentastomum denticulatum have been found in rare instances. The ascaris lumbricoides is sometimes found, usually after death, in cases of peritonitis from perforation. There is no satisfactory evidence that it can perforate the intestinal wall, but it easily makes its way through an existing hole. A certain interest is to be attached to the discovery by Winckel of the filaria sanguinis hominis in the milky fluid withdrawn from the abdomen of a patient who had lived in Surinam.

The echinococcus when present in the peritoneal cavity is usually also found elsewhere, especially in the liver. Hundreds of cysts may be present in the omentum and mesentery, forming tumors of large size. Numerous cases of echinococci in the pelvis have been reported. In one instance two hundred cysts are reported to have been spontaneously discharged through the peritoneum. The disturbances resulting from their presence are due chiefly to the size of the tumor. This may be large enough to interfere with respiration or to disturb digestion. In the pelvis they have been known to interfere with childbirth and to cause retention of urine and constipation. As a rule, the resulting tumor increases slowly, without pain or fever and with no disturbance of the general health. Rupture of the cysts and discharge of the fluid into the peritoneal cavity have been followed in a number of instances by peritonitis, and in still other instances by urticaria. Perforation into the intestine or vagina may be followed by extensive suppuration, and death from septicæmia.

The tumor is rounded, elastic, fluctuating, and at times gives rise to a thrill on percussion, suggesting the quivering of jelly. The aspirated fluid is pale, transparent, of a specific gravity of 1006 to 1010. It contains a faint trace of albumin, grape-sugar in minute quantity, and abundant chloride of sodium. Hooklets, scolices, or portion of the cyst-wall may be found in the sediment.

Diagnosis.—The tumor may be mistaken for other abdominal tumors and for ascites. It resembles ovarian and parovarian cysts in its slowness of growth and absence of characteristic symptoms. As a pelvic tumor it rarely attains the size which may be reached by these, and if omental or mesenteric it occupies the upper abdomen at the onset. In this respect it resembles the pancreatic cysts, which usually have a more distinctive method of origin, and are associated with greater digestive disturbance and discomfort. The aspirated fluid may be absolutely characteristic; the thrill is less significant, is often lacking, and may be present in ascites and in ovarian tumors.

Treatment.—The medical treatment of intraperitoneal echinococcus ceases with the establishment of the diagnosis. Until that time it consists merely in the attempt to relieve symptoms.

DISEASES OF THE LIVER.

BY REGINALD H. FITZ.

MALFORMATION.

DEFORMITIES of the liver are either congenital or acquired. The former is usually seen as the lobulated liver, which in most instances is to be attributed to hereditary syphilis. Whatever symptoms it may give rise to are those resulting from a chronic fibrous hepatitis, under which head they will be mentioned.

Corset-liver is the term applied to the acquired malformation of the liver most often found. It is the result of prolonged pressure from some sort of a waist-band. It is usually found in women, though sometimes in men accustomed to wear tight belts, as sailors.

A transverse furrow crosses the right lobe, dividing it into two unequal portions. In extreme cases the furrow is deep, and the compressed portion of liver is transformed into a fibrous band in which the liver cells have largely disappeared and the peritoneal surface is thickened. The smaller blood-vessels in this band are obliterated. The larger vessels are prominent, partly from the atrophy of the intervening parenchyma, partly from the dilatation of veins, lymphatics, and bile-ducts. The lower peripheral portion of the right lobe is likely to be rounded, and may be freely movable as if hinged. A coil of intestine may lie in the furrow between the liver and the abdominal wall.

There are no symptoms resulting from this deformity, unless it may somewhat obstruct the flow of bile from the gall-bladder, and thus favor the formation of gall-stone. Jaundice is rare in consequence of the deformity. Strümpell asserts that in extreme cases there may be a persistent sense of pressure and dragging in the hepatic region, and that severe pain, vomiting, and symptoms of collapse may follow recurring attacks of passive congestion of the separated portion of liver. This deformity is of chief importance in the suggestion it gives of an abdominal tumor, especially when its flatness is separated from that of the hepatic region by a tympanitic intestine.

The obvious treatment, if any be required, is the removal of pressure and the relief of congestive disturbances by rest on the back or side, and the application of heat.

Other acquired deformities of the liver may result from the pressure of tumors or of the ribs in spinal curvature: no characteristic symptoms are likely to result from such pressure.

MALPOSITION.

Displacements of the liver are either congenital or acquired. The former include the instances of transposed organs and of hernia of the liver through the anterior abdominal wall or through the diaphragm. In these cases there is no necessary disturbance of function.

Acquired displacements of the liver are those where the organ lies higher or lower than normal. The former results from the pressure of an abdominal lesion, ascitic fluid, or distended intestine; the latter is due to a pleuritic effusion, an emphysematous lung, an intrathoracic tumor, or a deformed thorax. The displacement which is most likely to be associated with symptoms of disturbance is the wandering or movable liver. This has been found chiefly among women, and especially among those in middle life who have borne children. Its occurrence is favored by a lax abdominal wall, muscular strain, and tight lacing. That the liver may become displaced it seems demanded that an abnormally large suspensory ligament, perhaps of congenital origin, should exist. Since the wandering liver has been recognized only during life, there is no evidence to indicate any other anatomical change than the elongated ligament.

The **symptoms** are those of tension and dragging, both increased on exertion; also of pain, sometimes severe, which may be referred to the right shoulder. The patient is apt to become nervous and hysterical.

The **diagnosis** is to be made by the recognition in the right half of the abdomen of a solid, unyielding, movable tumor, with its outlines corresponding to those of the liver. Palpation may indicate the fissure between the right and left lobes, and has suggested the furrows existing on the under surface.

The lower border of the tumor may be found in the right iliac fossa, and the upper border may be separated from the costal cartilages by a distinct interval in which the tense suspensory ligament has been felt. The region of normal hepatic dulness is occupied by a tympanitic area, and the abdominal tumor may be returned to the region normally occupied by the liver, with the return of the physical signs indicative of the presence of that organ.

The wandering liver may be mistaken for a cancerous omentum or for a tumor of the right kidney, ovary, or uterus, although the diagnosis has usually been easily made by detecting the characteristics above mentioned.

The **treatment** consists in a suitable bandage for preventing the displacement downward.

CONGESTION OF THE LIVER.

Congestion of the liver as a distinct disease scarcely deserves a place in modern works on the practice of medicine. The symptoms attributable to an active congestion of the liver are generally conceded to be the result of a gastro-duodenal catarrh or to a catarrh of the bile-ducts, and the anatomist finds no other evidence of an active congestion than that which might be the result of an obstructed outflow of blood.

The distinction is often drawn between the active congestion from increased portal inflow and a passive congestion from hindered venous outflow. This distinction is more theoretical than real, since it is difficult to conceive that the latter should occur without the former. If the former exists, its first effect should be to cause a distension of the radicles of the portal vein, the symptoms of which, as shown by the Goltz experiment, are wholly different from those attributed to active congestion of the liver.

Etiology.—It is customary, however, to admit the possibility of the occurrence of an active congestion of the liver from an increased influx of portal blood. This is recognized as a physiological process after meals, and may become pathological if the food be excessive in quantity or of an irritating quality (containing an excess of spice or alcohol), especially if the abundant eater be a person of sedentary habits. Other pathological causes of an active congestion of the liver are found in infectious diseases, especially in malaria, erysipelas, typhus and typhoid fevers, and in dysentery. Injury of the liver is regarded as a cause by many writers, and a vaso-motor disturbance is presented as an explanation of the symptoms attributed to congestion of the liver associated with suppression of the menstrual flow, either at a catamenial period or at the approach of the climacteric. If such symptoms are connected with uterine or ovarian irritation or with a suppressed hæmorrhoidal flow, they are also included under the vaso-motor congestion of the liver.

Morbid Anatomy.—An actively congested liver is one which should present the general characteristics of acute congestion—viz. increase in size, diminution of consistency, a dark reddish-blue color, and the escape of an increased quantity of blood on section, especially on pressure.

Symptoms.—There are no symptoms absolutely characteristic of an active congestion of the liver. Those which occur are essentially the same as in passive congestion, and are not materially different from those occurring in gastro-intestinal catarrh.

Diagnosis.—The diagnosis is based upon the recognition of an enlargement of the liver, with a sense of fulness and weight in the right hypochondrium, in connection with the conditions mentioned under Etiology.

Treatment.—A rational treatment for the immediate attack would consist in applying heat or cold to the region of the liver, perhaps leeches, and in giving a saline laxative. The diet should be liquid and farinaceous. To prevent further attacks, alcohol and spices should be avoided, food taken in moderation, but exercise freely. Malarial influences should be counteracted by quinine or removal to a non-malarial region. Hot vaginal douches and foot-baths should be employed to promote a catamenial flow.

PASSIVE CONGESTION OF THE LIVER.

Definition.—The result of mechanical obstruction to the outflow of blood from the liver.

Etiology.—Its causes are obstruction to the flow of blood through the heart in consequence of uncompensated valvular disease, or from a weakening of the myocardium by disease or degeneration of the muscle. Obstructed flow of blood through the lungs proves a cause in asthma, bronchitis, emphysema, fibrous pneumonia, atelectasis, and chronic pleurisy. The flow of blood may become obstructed through both heart and lungs by a deformed spine and by a considerable left-sided pleuritic effusion, thoracic aneurism, and mediastinal tumors. Aneurisms and glandular tumors may compress the inferior vena cava, and thus prevent the outflow of hepatic blood. Rarely the obstruction to the outflow may be the result of valves or projections from the wall of the hepatic vein or of constriction from surrounding inflammation.

Local causes of passive congestion may result from the pressure of tumors within the liver upon neighboring branches of the hepatic vein, or the latter may be obliterated by thrombi, or a passive congestion in part of the liver may take place below a constricted part, as in the corset liver.

Morbid Anatomy.—The anatomical changes in a liver from passive congestion become more conspicuous and serious the longer the mechanical obstruction has existed. At first the liver is enlarged symmetrically, the edges rounded, color purple, and consistency diminished. The capsule is smooth and shining, tense. Abundant blood flows from the cut surface, and the lobular regions are chiefly defined from the contrast between the dark purple of the central zones and the paler gray or yellow peripheral portion of the lobules.

The longer the congestion has existed, the smaller and denser the liver. The surface is then slightly roughened, the capsule opaque and wrinkled. The central zones are of a dark reddish-brown color, thin and unduly increased, while the portal region of the lobules is correspondingly diminished and is represented by highly elevated gray or yellow lines and spots—the atrophic nutmeg liver. The radicles of the portal vein in the gastro-intestinal mucous membrane are dilated; the spleen is somewhat enlarged, dense, and of a dark-purple color. The pancreas is denser and darker than normal, and the kidneys are usually dense and purple, since the causes preventing the outflow of blood from the liver are likely to produce the same result in the kidney.

Symptomatology.—Symptoms directly referred to the liver only become apparent when the congestion has existed for some time. There is then a sense of weight and fulness in the right hypochondrium on sitting up or lying on the left side. The liver, if enlarged, may be tender on palpation. The breath may be short from obstruction to the descent of the diaphragm, and pain may extend from the liver to the right shoulder. The short breath and pain are likely to occur at intervals in consequence of temporary excessive use of the heart and lungs.

Jaundice is usually slight, often noticeable only in the conjunctivæ and face. The urine may be free from bile-pigment, and the stools of normal color. When cardiac obstruction is the cause of the hepatic congestion, the jaundice is more conspicuous, and the face may present a greenish tint from the combination of jaundice and cyanosis. In the cardiac cases the frequently associated gastro-duodemal catarrh may cause a marked jaundice, and this catarrh is to be regarded as an important cause of the frequent loss of appetite, nausea, vomiting, belching, and epigastric pain which may be present.

Ascites becomes a symptom of the later stages of the atrophic congested liver. Its degree is usually in proportion to the general œdema.

The physical examination of the liver in the earlier stages of chronic passive congestion gives evidence of enlargement of the organ, the lower edge of which may be found below the navel. The degree of enlargement may vary materially within a short time, excessive exertion being a cause for a rapid increase in size, which quickly subsides after rest. On inspection there sometimes may be seen a greater fulness of the right hypochondrium, and epigastric pulsation becomes conspicuous when the tricuspid valve is incompetent.

Verstraeten calls attention to the importance of auscultation in determining the site of the lower border of the liver. If the patient lies on the back, with enlarged abdominal muscles, the heart-sounds may be readily heard with the stethoscope over the region of the liver, becoming suddenly feeble at the lower border. They are easily transmitted, rendered feeble or interfered with by a layer of lung between the heart and liver. A tense abdominal wall conducts the sounds beyound the hepatic area. Percussion will usually give an increased area of liver dulness below the right costal cartilage and in the epigastrium.

The degree of enlargement is less readily determined by percussion than by palpation, since the intestinal resonance frequently obscures the hepatic dulness. The lower edge of the liver is usually to be distinctly felt by the finger-tips of the right hand, especially if strong pressure upward in the right lumbar region be made by the left hand. The lower edge of the liver is then to be felt as a dense, smooth, sharply-defined mass, changing its position slightly on respiration. The notch between the right and left lobes is sometimes to be felt, especially where deepened.

The atrophied congested liver, on the contrary, is usually so much diminished in size as to escape the physical methods of determination.

Diagnosis.—Passive congestion of the liver is to be suspected with the recognition of the causes which may give rise to it. If the liver is then found to be enlarged, and especially if subject to rapid variations in size, the diagnosis is plain. If the causes are recognized and the liver be not enlarged, an atrophic congested liver may be diagnosticated in the presence of the symptoms mentioned, especially if anasarca have preceded the ascites and the latter be not extreme.

Prognosis.—The existence of passive congestion of the liver is evidence of persistent, progressive, mechanical obstruction to the circulation, the effects of which may be palliated, but the causes of which are generally irremediable.

Treatment.—The indications for treatment are to preserve the strength of the patient by easily-digested nutritious diet (fresh meat, fish, eggs, milk, fruit, vegetables, and farinaceous food). The force of the heart is to be strengthened by digitalis, strophanthus, and strychnine, while the portal vessels are to be depleted by means of vegetable laxatives, as rhubarb, senna, aloes, podophyllin, colocynth, and jalap, or by the salines, as Epsom salts, Crab Orchard salts, Hawthorn, Geyser, Friedrichshall, or Hunyadi waters. Occasional doses of calomel or blue pill are preferred by some patients. If ascites be present, it may be treated as mentioned in the consideration of that subject.

PERIHEPATITIS; SUBPHRENIC ABSCESS; SUBPHRENIC PYOPNEUMOTHORAX.

Etiology.—Inflammation of the peritoneal covering of the liver is either part of a general peritonitis or it is a peritonitis circumscribed to the immediate vicinity of the liver. The latter is usually secondary to a pathological process elsewhere.

The acute, suppurative variety may be caused by direct violence, as from a blow, but it is much more often the result of a perforating ulcer of the stomach or duodenum, abscess, or echinococcus of the liver, suppurating bile-ducts, perforation of the gall-bladder, abscess of the right kidney, and suppurative paranephritis or paracolitis in the course of an appendicitis.

The chronic fibrous variety represents the termination of the acute form, or it is the result of a chronic irritant, as tight corsets or straps, prolonged pressure, as in certain trades, subjacent cancerous or gummous nodules, an obstructed gall-bladder, or a continued pleurisy. Although usually seated over the right lobe of the liver, it may be limited to the vicinity of the left lobe, in which case embolic or traumatic abscess of the spleen or rupture of that organ is usually found to be the cause.

Morbid Anatomy.—In acute perihepatitis the peritoneum of the liver and of the corresponding under surface of the diaphragm is injected, dull, opaque, covered with a layer of fibrin. The opposed peritoneal surfaces are adherent to a greater or less extent, or the adhesions wall off from the general peritoneal cavity the space between the diaphragm and the liver, which may contain a quart or more of pus (subphrenic abscess), or the pus may be mixed with air or gas (subphrenic pyopneumothorax). The pus may be of a yellow-ochre color, and contain bilirubin crystals (Leyden), due to the admixture of pus and bile, or it may be fatty degenerated and contain crystalline fatty acids. These changes are usually found between the right lobe of the liver and the diaphragm, but they may be present over the left lobe when the cause of the perihepatitis lies to the left of the suspensory ligament.

In chronic fibrinous perihepatitis the peritoneum covering the liver is thickened, dense, and opaque over large and small areas. Fibrous adhesions unite the opposed surfaces as bands between the liver and the diaphragm, stomach, colon, or anterior wall of the abdomen. The fibrous thickening of the capsule may be present near the portal fissure. The shrinkage of the fibrous tissue may produce a globular or lobulated atrophy of the liver, and may give rise to narrowing or obliteration of the portal or hepatic veins, or of the cystic or common bile-ducts.

Symptoms.—At the outset of an acute perihepatitis the symptoms are usually violent, and are suggestive of a perforating peritonitis circumscribed to the region of the liver. Severe pain and tenderness are experienced in the epigastrium or right hypochondrium, increased both on prolonged inspiration and on pressure; a continued fever, sometimes preceded by a chill, follows. The breathing becomes increased in rapidity, and there may be slight jaundice. Loss of appetite, nausea, and perhaps vomiting, are likely to occur.

On physical examination the signs closely simulate those due to a pleuritic effusion. The right hypochondrium is usually distended, the intercostal spaces are motionless. There is dulness on percussion, perhaps as high as the inferior angle of the scapula, and in the dull area there is an absence of voice and of respiratory sounds. At the outset a friction sound may be felt or heard over the liver, but this usually quickly disappears.

The lower edge of the liver may lie on a level with the navel. It has been observed that the lower border of hepatic dulness was replaced by a tympanitic area when the patient lay on the left side, suggesting rather a movable than an enlarged liver.

The **course** of an acute perihepatitis may be rapid, terminating in recovery in the course of a few days, or, in case of suppuration, may be prolonged throughout weeks or months. In the latter case prolonged and irregular elevation of temperature, progressive loss of flesh and strength, suggest deep-seated suppuration. The abscess may perforate the diaphragm and the pus be evacuated into the lung, or the abscess may break into the stomach or intestine or through the abdominal wall. In the course of this affection thrombosis of the hepatic vein or of the inferior vena cava may take place, in the latter instance with resulting œdema of the legs. In general the course of subphrenic abscess simulates that of an empyema or an abscess of the liver, and the prognosis is always doubtful unless efficient drainage of the abscess be established. In those instances where the pus is absorbed or evacuated through the lungs or gastro-intestinal tract dense cicatricial tissue may form in the wall of the abscess. By its contraction pressure may be produced upon the common and cystic ducts or upon the hepatic or portal veins, resulting in persistent jaundice or in passive congestion of the liver and ascites. The compression of the liver may be such that extreme degrees of atrophy may follow.

Diagnosis.—The rational and physical signs are suggestive of a pleuritic exudation, but the antecedents are indicative of a perforation of the stomach or duodenum or gall-bladder. The differential diagnosis lies between an empyema or pneumothorax above or below the diaphragm. The absence of cough, expectoration, and displacement of the heart negatives a pleuritic process. The depression of the lower border of the liver and the bulging of the right hypochondrium are usually greater than in pleuritic exudation. Especial value in differential diagnosis is to be attached to the results of an exploratory puncture. This should be made in the seventh or eighth interspace in the axillary line. Pfuhl has called attention to the spurting of the fluid through the trocar on inspiration, owing to the descent of the diaphragm, while in pleurisy the fluid spurts on expiration. The presence of bile-pigment in the pus would give further evidence of the seat of the abscess below the diaphragm.

Treatment.—In the earlier stages of acute perihepatitis the treatment should be directed to the relief of pain by the application of sinapisms, stupes, poultices, and leeches, and by the internal administration of sufficient doses of morphine. With the eventual recognition of pus the treatment becomes surgical, and demands an opening of a size sufficient to ensure drainage, perhaps necessitating the resection of a portion of one of the lower ribs. As much as a quart of pus has escaped immediately after an opening has been made.

Chronic fibrous perihepatitis is usually recognized after death, there being no characteristic symptoms. Except when it is the terminal condition of a subphrenic abscess, its importance is slight, and it represents a trivial complication of the pathological process to which it owes its origin.

ACUTE PARENCHYMATOUS HEPATITIS; ACUTE YELLOW ATROPHY OF THE LIVER.

Idiopathic acute yellow atrophy of the liver is a disease to be distinctly separated from the secondary acute atrophy that may serve to close the course of an obstructive jaundice or of a fibrous hepatitis.

Etiology.—It is one of the rarest affection of the liver. Thierfelder in 1878 estimated that about 200 cases of this disease were referred to at greater or less length in medical literature. Riess, however, saw 5 cases in three months, and Arnold saw 4 fatal cases among ten diseased soldiers during the course of three months—facts which have suggested possible epidemic influence. It occurs in women rather more frequently than in men, and its presence in pregnancy, usually in the latter half, is noteworthy. Although more frequently found in the prime of life, cases have been reported in infancy, childhood, and old age. Intense mental excitement and alcoholic excess have been deemed exciting causes, and its occasional occurrence in the course of typhoid and relapsing fevers, diphtheria, pyæmia, and septicæmia has been considered significant. The similarity of the symptoms has suggested phosphorus-poisoning as a cause, and the course and lesions have led to the theory of an infectious origin, which has derived a certain support from the repeated discovery of bacteria in the diseased liver.

Morbid Anatomy.—The liver is almost invariably diminished in size, perhaps two-thirds or three-fourths, though claimed to be slightly enlarged at the outset, and becomes flattened, cake-like, from a predominant shortening of its vertical diameter. The color is of a dirty yellow, and the consistency firm. The capsule is wrinkled, and the liver is flabby when handled.

On section the color may be uniformly yellow and opaque, the lobules indistinct, or there may be alternations of irregular patches of red and yellow. The red patches represent those portions of the liver from which the fat has been absorbed. After the liver has been exposed to the air for some time the cut surface will often show small white specks, crystals of leucin and tyrosin. The microscopic examination of the liver shows an extensive and extreme fatty degeneration of the liver-cells, a slight degree of cell-infiltration of the interstitial tissue, and scattered crystals of leucin, tyrosin, and bilrubin. Clumps of cells indicative of new-formed bile-ducts or bands of liver-cells are also to be seen.

In addition to the general yellow discoloration of the tissues of the body, there is found a fatty degeneration of the renal epithelium, of the heart, and of the voluntary muscles. The spleen is hyperplastic; numerous small hæmorrhages are found in various parts of the body and evidences of catarrh are present in the mouth and intestine. An excess of fluid is likely to be found in the pleural and pericardial cavities.

Symptoms.—The characteristic symptoms of acute yellow atrophy of the liver are present, as a rule, only two or three days before death. For a week or two previous to their onset, however, the patient suffers from the group of symptoms usually attributed to a gastro-duodenal catarrh. These are loss of appetite, nausea, vomiting, belching, irregular stools, tender epigastrium, headache, and prostration, as a rule. Eventually, a slight degree of jaundice appears.

With a rapid increase in the intensity of the jaundice the symptoms suddenly become more severe. The vomiting becomes constant—at first of the bile-stained contents of the stomach, then of a bloody fluid. The patient becomes nervous and restless. His headache becomes intense, until a delirium, almost maniacal, sets in. This is associated with general or localized convulsions. The patient becomes stupid, and eventually comatose.

During this period hæmorrhages, nasal, gastro-intestinal, urinary, or cutaneous, may occur. The pregnant woman is likely to abort, and the flowing may become excessive. There is an absence of fever until just before death, when the temperature may rise to upwards of 104° F.: on the contrary, it may become subnormal at that time. The pulse, which is rather slower at the outset, becomes variable, and is eventually rapid and feeble.

With the onset of the severe symptoms the area of hepatic dulness diminishes, and may soon wholly disappear. The region of the liver becomes morbidly tender, apparent (according to Riess) even in profound coma. An enlargement of the spleen is usually perceptible.

The urine is bile-stained, and its quantity becomes markedly diminished, complete suppression sometimes taking place. It is acid, its specific gravity varies between 1012 and 1030, and it contains a moderate quantity of albumin, in addition to bile-pigment and bile-acids. Hyaline and fatty (epithelial) casts are found. The great diminution, or even total absence, of urea is an especial characteristic. In its place leucin, tyrosin, and other products of retrograde metamorphosis are found. The presence of the former, determined by the evaporation of a small quantity of urine to which acetic acid has been added, is of especial value in diagnosis. Leucin and tyrosin, however, although usually, are not invariably, present.

Diagnosis.—The diagnosis of acute yellow atrophy becomes possible only with the appearance of the grave symptoms. The sudden onset of restlessness, delirium, and convulsions, fever being absent, in a case of jaundice, should at once arouse suspicion. The presence of leucin and tyrosin in the urine, and the progressive diminution in the size of the liver, confirm the diagnosis. Similar symptoms may occur at the end of a case of hypertrophic cirrhosis, but the long duration of this affection and the enlargement of the liver are sufficient to exclude this affection.

Greater difficulty may be experienced in the differentiation of phosphorus-poisoning. In this affection the gastro-enteric symptoms are more severe, the jaundice and profound nervous symptoms are of earlier occurrence, and leucin and tyrosin are usually absent from the urine. The liver is enlarged for some time; despite the severity of the symptoms, it is exceptional for it to be found atrophied, and the diminution in size does not take place so rapidly as in acute yellow atrophy.

Prognosis.—The disease is fatal, death usually occurring in the course of a fortnight from the onset of the symptoms, although more chronic cases are recorded where death did not occur for a couple of months. Instances of recovery have been reported, but the diagnosis in these cases is always to be doubted.

Treatment.—There is no other indication for treatment than the relief of symptoms. Ice by the mouth and morphine subcutaneously should be used to check vomiting. Cold applications may be made to the head, and opium and chloral may be given to relieve the nervous symptoms. Stimulants are to be employed in the stage of collapse.

SUPPURATIVE HEPATITIS ; ABSCESS OF THE LIVER.

Etiology.—Abscesses are formed in the liver in consequence of the introduction into its substance of a pyogenic, usually infectious, irritant. Such an irritant may enter the liver directly, as in wounds of this organ, or it may be admitted through the blood-vessels or by means of the bile-ducts. This irritant is usually bacterial or amœbic, although the evidence in favor of a chemical irritant as a cause for certain hepatic abscesses is not to be overcome. Traumatism produces an abscess when its action is combined with that of an infective agent, and the occurrence of abscess of the liver after injury to the head or extremities, especially in the presence of open wounds or osteo-myelitis is thus explained. The injured liver suppurates because it becomes invaded by the pyogenic irritant. Dabney's study gives evidence of the rarity of the origin of abscess of the liver from disease of the bones or parts of the body other than those intimately connected with the liver.

The physician is chiefly concerned with those cases of hepatic abscess which proceed from the blood-vessels or the bile-ducts. The former include the embolic abscesses of the liver, especially those resulting from embolism of the portal vein ; also those due to the extension of a pylephlebitis into the liver. The source of these is to be sought for in an inflammation affecting the radicles of the portal vein, especially the inflammatory and ulcerative processes of the large bowel found in dysentery, appendicitis, and piles. Despite the frequency of ulcers of the intestine in typhoid fever, abscess of the liver is an unimportant complication of this disease. An umbilical phlebitis may form the source of abscess of the liver in the new-born, and primary abscess of the spleen may serve as a source of hepatic abscess by the transfer of emboli through the splenic branch of the portal vein, or by the continuance of a phlebitis along its course from the spleen to the portal vein, and thence into the liver.

Embolism of the hepatic artery is an infrequent cause of hepatic abscess, but will explain the occurrence of the latter in ulcerative endocarditis and in gangrene of the lung. A bland embolus carried through the hepatic artery may serve as the exciting cause of an abscess of the liver, as suggested by Osler and Ross, the embolus being carried from an aneurism of the hepatic artery to parts of the liver where the influence of pyogenic agents becomes superadded. Regurgitant embolism of the hepatic vein is a possible cause of hepatic abscess when a septic thrombosis is present in the peripheral venous system.

Suppurative inflammation of the bile-ducts (cholangitis) is a not infrequent cause of abscess of the liver, and in many instances represents the extension of a gastro-duodenal catarrh along the common bile-duct into the hepatic ducts. An abscess of the liver is the more likely to result if gall-stones, parasites (echinococci, lumbrici, distoma), or foreign bodies (needles, pins, nails) are present in the bile-ducts.

Although hepatic abscesses occur with greater frequency in the tropics than in the temperate zones, it is doubtful if this fact indicates any difference in the etiology. The high liver is usually the male white, although Moore says that natives are as liable to hepatic abscess as Europeans ; and his frequent bilious attacks present the characteristics of a gastro-duodenal catarrh, whose tendency to produce a catarrh of the bile-ducts has already been suggested. This and the frequency of dysentery in the tropics—for Kiener and Kelsch found that dysentery and hepatic abscess concurred in 85.35 per cent. of 314 cases—offer the most satisfactory explanation of the origin of the tropical abscess, especially since the relation of the amœba coli to dysentery and abscess of the liver has been made so clear through the researches of Kartulis, Councilman and others.

Morbid Anatomy.—The liver of suppurative hepatitis is usually symmetrically enlarged, and may on inspection present no abnormal appearances suggesting the presence of pus. One or more circumscribed, yielding, opaque, yellowish-white, circular patches, coated with a fibrous layer, often give superficial evidence of the abscess to be found on section of the organ.

The abscesses vary somewhat in appearance, as they are single or multiple, although the single abscesses often, if not usually, result from the confluence of many. The single abscess is limited to the right lobe of the liver, according to Waring's figures, in two-thirds of the cases, is usually near the convexity, and may be as large as a child's head, containing several quarts of pus. The wall is usually shreddy, not sharply defined from the surrounding injected liver-tissue except in the more chronic cases, when it may be circumscribed, thick, dense, grayish-white, suggestive of cartilage. The contents are rather puriform than purulent; an opaque fluid of viscid or creamy consistency, yellow or red, forming an emulsion of minute granules, and containing fat-drops (free and in large cells), also leucocytes and innumerable minute rhombic or clustered acicular crystals of bilirubin (hæmatoidin). The presence of amœbæ or hooklets in the contents of these abscesses gives evidence of their parasitic origin.

The multiple abscesses may be fifty or more, scattered throughout the liver, though often grouped in clusters. They vary in size, usually from that of a pea to that of a plum, the larger being irregularly rounded and trabeculated as if formed by the confluence of the smaller. The walls are usually shreddy, the contents viscid, odorless or fetid, yellow or bile-stained. The presence of puriform thrombi in the branches of the portal vein or of arborescent patches of necrosis indicates the vascular origin of the abscess, while abundant staining with bile, and especially the presence of dilated bile-ducts with purulent contents, gives evidence of the relation of the abscess to a cholangitis.

When the single or multiple abscess reaches the surface of the liver, the pus may escape into the peritoneal cavity, or, an adhesive peritonitis having previously taken place, may pass into a neighboring hollow organ, as the stomach, colon, duodenum or pelvis of the right kidney. Perforation of the diaphragm is not infrequent, with the passage of the pus into the lungs and bronchi. The pus may, however, enter directly the pleural or pericardial cavities or pass through the abdominal wall, escaping near the liver, or, turning upward, downward, or backward, make its appearance at some distant point.

In rare instances the pus may become inspissated, caseous in appearance, perhaps calcified and encapsuled in dense fibrous tissue. The abscess may also become transformed into a cyst-like cavity, with more or less viscid colorless contents, in which plates of cholesterin may be found.

Symptomatology.—The symptoms of suppurative hepatitis may be so little characteristic that the abscesses are unexpectedly found after death, or are first suspected after the evacuation of the pus through the bowels or bronchi. Small abscesses may produce no symptoms; the course of large abscesses may be latent, and the symptoms of the suppurative hepatitis may be wholly obscured by those of the disease giving rise to the hepatic abscess.

The general symptoms are the progressive loss of flesh and strength, associated with failing appetite, nausea, vomiting, and a sense of weight and fulness in the epigastrium. The association of these symptoms with a dry cough and hectic have not infrequently led to a mistaken diagnosis of pulmonary phthisis.

The stools are irregular; either diarrhœa or constipation may be present. The movements become suddenly liquid when the abscess empties into the bowel, and the evacuated pus may be found to contain large quantities of amœbæ. Toward the end of the disease, in fatal cases, nervous symptoms become conspicuous, as wakefulness and delirium, or stupor ending in coma.

Most important as suggesting the presence of suppuration is the range of temperature. In the chronic abscess of the liver pursuing a latent course the elevation of temperature may be so slight as to be in no way characteristic. As a rule, however, considerable degrees of elevation are to be found, as high even as 104° or 105° F. These elevations may follow chills of greater or less severity, and be followed by defervescence with sweatings, the latter sometimes very profuse, with such regularity as to suggest the progress of a malarial fever, but the intervals between the chills become very irregular in the course of time. In other cases a continued elevation of temperature, with greater evening rise, may be present, suggesting rather the course of a typhoid fever. The temperature may fall to the normal with the evacuation of the pus, or become permanently lowered toward the end of life. The rate of pulse corresponds in general to the temperature, rising and falling with it, but becomes markedly frequent, high, and feeble as death is neared.

The frequency of the respiration follows the course of the temperature and pulse in those cases where there is no considerable enlargement of the liver. In the latter event it becomes increased from the elevation of the diaphragm, and painful when the serous covering of the diaphragm becomes inflamed. A spasmodic dry cough is then likely to be present. A certain degree of dyspnœa is frequent from the collapsed condition of the right lung and the bronchial catarrh which result from the diminished air-space.

The local symptoms which suggest the liver as the seat of the suppuration are the enlargement of this organ and the pain which may proceed from it. Of these the enlargement is the more constant, and may be more apparent when the patient is upright then when he is lying down. It is due, in part, to the quantity of pus formed in the abscess, in part to the parenchymatous changes in the liver-cells, and in part to the congested state of the organ. The liver is symmetrically enlarged until the abscess projects above the surface, and its size is usually in proportion to the volume of the contained abscess or abscesses. Parts of the liver may be honeycombed with pus-cavities, and the enlargement be slight; on the contrary, the upper border of hepatic dulness may extend to the second rib in front and to the spine of the scapula behind, while the anterior edge of the right lobe may be on a level with the crest of the ilium. Since the larger abscesses usually project from the upper surface of the right lobe, the area of thoracic dulness is frequently sharply convex upward.

The collapsed lung is made evident by the dulness on percussion, the bronchial breathing, and the increased vocal resonance in the affected region.

The liver is readily palpated when it projects below the costal edge, and a palpable or audible peritoneal friction may be recognized in case the peritoneum over the abscess becomes inflamed. In rare instances palpation may reveal increased elasticity or a sense of fluctuation at the seat of the abscess.

Localized pain is a most important symptom, as suggesting the liver as the seat of the pus. It does not occur unless the suppuration nears the surface, which may take place either early or late in its history. The pain may be deep-seated and extend to the right shoulder when the branches of the phrenic nerve situated in the capsule of the liver and in the suspensory ligament are irritated, since they arise from the fourth cervical nerve, which also supplies the shoulder to which the pain becomes referred through the central nervous system (Luschka). The pain may be more superficial, and associated with tenderness and a tense rectus, when the anterior surface of the liver is the seat of a localized peritonitis. Because of local pain the patient usually finds his most comfortable position on the back or on the right side.

Slight degrees of jaundice are not infrequent, and may come and go with the exacerbation and remission of the febrile temperature. More considerable and more prolonged jaundice may result from the pressure of the abscess on the bile-ducts. Ascites from pressure of the abscess upon the branches of the portal vein, or anasarca from pressure of the enlarged liver upon the inferior cava, are rarely noticed.

Moderate enlargement of the spleen is not infrequent in the acuter varieties of suppurative hepatitis, but is the result rather of hyperplasia than of obstructed venous outflow.

Suppurative hepatitis is usually a chronic affection, lasting months or years. It may terminate fatally in the course of a fortnight or the patient may live for five years (Ewald).

Since the abscess formation is often multiple at the outset, with a tendency to coalescence, the longer the suppurative hepatitis exists the more likely is the single abscess to form. As this becomes larger it tends to break into the peritoneal cavity, producing a fatal peritonitis, or into the stomach or intestine, lungs and bronchi, into the right renal pelvis, or through the external abdominal wall, with a lowering of the temperature and an alleviation of the symptoms. The discharge into the respiratory tract is usually of a reddish color, suggesting anchovy sauce (Osler), and may be found to contain amœbæ. The fistulæ thus established may close and recovery occur, or they may remain open, death resulting from exhaustion. As a rare complication are associated abscesses in the brain.

Prognosis.—Suppurative hepatitis is a grave malady. Single small abscesses may perhaps be recovered from by the inspissation and calcification, or by the softening and absorption, of the pus. Multiple small abscesses are usually fatal, since they are inaccessible to surgical treatment unless the patient's strength endures until they coalesce. As a rule, unless the pus be evacuated spontaneously or by the surgeon, death occurs. Even when the pus is evacuated and the patient apparently improves, a fatal result may occur from hæmorrhage, embolism, peritonitis, or septicæmia, and, although with the evacuation of the pus the prognosis becomes more favorable, a permanent fistula may arise, and death eventually takes place from amyloid disease. The usual duration of the disease in fatal cases is from six weeks to three months. In cases not treated surgically the mortality may be as high as 80 per cent., while in cases operated upon there may be only 30 per cent. of deaths. Perforation of the abscess into the lung or into the gastro-intestinal tract resulted favorably in at least 50 per cent. of the cases treated by Ronis.

Diagnosis.—A diagnosis of suppurative hepatitis is always doubtful unless an abscess of considerable size has formed or the aspirator shows the presence of pus. It depends upon an appreciation of the existence of the causes of suppurative hepatitis, especially residence in the tropics, dysentery, ulcerative processes in the gastro-intestinal tract, upon the presence of a continued or intermittent fever with or without chills, and a painful and tender enlargement of the liver, a part of which may show an extreme degree of elasticity, perhaps actual fluctuation.

Suppurative hepatitis is often confounded with malarial fever. In the former quinine gives no relief, the splenic enlargement is not so extreme, and there are no malarial organisms in the blood.

Recurring attacks of hepatic colic from gall-stones in the common or hepatic ducts also closely simulate suppurative hepatitis. The pain is usually more intense, the jaundice greater and more constant, and the febrile attack brief. As the abscess attains a large size, it may be mistaken for an echinococcus cyst, which develops more slowly and without fever. It may also resemble an empyema from the long duration of the fever and the physical signs. Usually in abscess of the liver the dulness is higher in front than behind, although the level may be the same. In empyema the retracted lung lies along the spine and at the upper part of the thoracic cavity, while the lung is everywhere in intimate relation with the subphrenic abscess of the liver.

The use of the aspirator eventually becomes necessary to establish the diagnosis, and a negative result does not exclude suppurative hepatitis, since the pus may so lie as not to be reached by the point of the needle, or be so thick as not to flow through its canal. The patient should be anæsthetized, since several punctures may be necessary, and the needle should be used only to make clear the need of the knife. Puncture should be made into tender or yielding spots in the enlarged liver below the costal cartilages, and into the seventh right intercostal space in the axillary line, or at this level in front or behind in case the region of dulness extends above this point. Puncture is not absolutely harmless, and resulting hæmorrhages may be profuse while the needle remains in the liver.

If a spontaneous discharge of pus has taken place through the lungs or bowels, the presence of amœbæ, liver-cells, or bile-pigment may indicate its source, and the presence of hooklets give evidence of its relation to echinococci.

Treatment.—The only medicinal treatment of suppurative hepatitis is to relieve symptoms and sustain strength until the abscess has reached such a size as to be accessible to the surgeon or finds an outlet through the lungs, stomach, or intestine. The pain may be relieved by the application of heat, cold, sinapisms, or dry cups over the sensitive liver. The conjoined use of morphine may be necessary. Mild salines, as Carlsbad and Rochelle salts, or citrate of magnesium, phosphate of sodium, or chloride of ammonium, may be used to promote evacuation of the bowels. Rhubarb, euonymin, or iridin may be given at bed-time.

The diet should be largely liquid and unirritating—whey, milk (with lime-water or soda-water if desirable), beef-juice and milk, gruels, and toasted bread.

The use of leeches, blisters, mercurials, cholagogues nitro-muriatic acid, so often recommended for the purpose of preventing the formation of pus in acute hepatitis, was based essentially upon erroneous theories of inflammation and the origin of pus. There is no reason to suppose that they are of the slightest use in preventing its formation.

FIBROUS HEPATITIS; CHRONIC INTERSTITIAL HEPATITIS; CIRRHOSIS.

Etiology.—Fibrous hepatitis may be regarded as the result of irritants brought to the liver by means of the blood-vessels, especially the portal vein. or through the bile-ducts, or by an invasion of the peritoneal capsule. The immediate cause, in nearly two-thirds of all cases, is alcohol, especially that variety containing fusel oil, obtained from the fermentation of grains and potatoes. The influence of excessive drinking of strong liquors has led to the use of the term "drunkard's liver" or "gin-drinker's liver" as synonymous with fibrous hepatitis. All drunkards are not sufferers from this affection, gin is not the liquor principally concerned, and fibrous hepatitis is often found in alcoholic patients who would not come under the class of drunkards. Rarely the excessive use of stronger wines and beer may produce fibrous hepatitis.

It has been claimed, partly as the result of experiments on animals, that spices, coffee, alkaloids (ptomaïnes), phosphorus, arsenic, and antimony, may be brought to the liver from the intestines through the portal vein, and produce fibrous hepatitis. Satisfactory clinical evidence of the truth of this view is lacking.

Certain acute infectious diseases—in particular intermittent, typhoid, and scarlet fevers, cholera, and dysentery—are claimed as occasional causes of fibrous hepatitis by contaminating the blood.

Among chronic infectious diseases syphilis holds high rank as a cause of fibrous hepatitis. An increase of the fibrous tissues of the liver is to be recognized as an accompaniment of miliary tuberculosis, but produces no symptoms by which its presence can be recognized during life. Fibrous hepatitis has been found as an accompaniment of rickets, and Murchison considers that it may result from gout. Eichhorst is inclined to recognize a senile variety, analogous to the arterio-sclerotic nephritis resulting from senile changes in the blood-vessels.

Evidence of the origin of a fibrous hepatitis from the transfer of a visible mechanical irritant to the liver is given by Welch in his description of the pigmented liver of coal-miners.

When obliteration of the portal vein is associated with fibrous hepatitis, it is generally regarded as a result of the latter, although Botkin claims that it may be a cause, basing his claim on Solovieff's experiments.

The obstruction to the outflow of venous blood from the liver, often included among the vascular causes of fibrous hepatitis, has already received sufficient consideration in the section upon Congestion of the Liver.

The biliary causes of fibrous hepatitis are those producing chronic inflammation of the bile-ducts. These are gall-stones, tuberculosis, or congenital disturbances producing obstruction, closure, or obliteration of the bile-ducts.

Fibrous hepatitis sometimes results from the extension of a chronic perihepatitis into the substance of the liver. This perihepatitis is usually part of a chronic peritonitis.

Fibrous hepatitis occurs oftener in men than in women, and usually between the ages of thirty and sixty years. Howard has collected 63 cases among children before puberty, and Hatfield adds 93 to this list. It has been found in the new-born and in the fœtus.

Morbid Anatomy.—The essential feature of fibrous hepatitis is the excessive increase of fibrous tissue in the liver. This fibrous tissue usually tends to shrink. It is claimed by some that the new-formation of fibrous tissue proceeds from the interstitial tissue of the liver, and that the liver-cells become secondarily affected, while others maintain that the changes begin as a degeneration and destruction of the liver-cells, and are followed by the interstitial changes.

Of greater importance perhaps is the question which has been raised as to the origin of the increase of fibrous tissue in the vicinity of the blood-vessels or of the bile-ducts. Charcot and Gombault, in particular, discriminate between an insular cirrhosis the result of an irritation proceeding from the bile-ducts, an annular cirrhosis representing an outgrowth of fibrous tissue around the veins, and a monocellular cirrhosis in which the growth of fibrous tissue surrounds the separate liver-cells.

With this anatomical distinction was associated a difference in etiology, it being claimed that annular cirrhosis was the result of alcohol, insular cirrhosis of the inflamed bile-ducts, and monocellular cirrhosis of syphilis. These distinctions are not generally accepted.

Still more important, clinically at least, is the distinction which must be drawn between hypertrophic cirrhosis and atrophic cirrhosis. The former is represented by a liver which is symmetrically enlarged, perhaps doubled in size, weighing as much as eight pounds, while the latter term is applied to a liver which may be reduced in size at least two-thirds, weighing only a pound. In both there is an increased quantity of fibrous tissue. In hypertrophic fibrous hepatitis there is often, not invariably, an abundant fatty infiltration of the liver-cells, and the hypertrophic cirrhosis may then represent this combination. In the absence of such fat-infiltration the hypertrophied fibrous liver has been regarded by Ackerman as analogous to the condition of the mammary gland in diffuse fibroma, the so-called chronic mammary hypertrophy.

The anatomical changes usually presented in chronic fibrous hepatitis are generally spoken of as representing two stages—the one of enlargement, the other of atrophy. This distinction is of no especial value, as the enlarged liver is usually not particularly enlarged, and the functional disturbances are not apparent until atrophy takes place.

The longer the process continues the smaller is the liver, until it may be reduced to one-third of the normal size. Its shape is the more deformed the greater the atrophy. The surface is covered with granules and nodules, varying in size from that of a pin-head to that of a walnut and upward; hence the terms "granular liver," "hob-nailed liver," and "lobulated liver." The left lobe may be so shrunken as to form a mere band or annular appendage. The density of the liver is increased in proportion to the degree of atrophy, and becomes tough and leathery. The cut surface is of a reddish-gray or grayish-yellow color, corresponding to that of the surface, and is studded with the sections of granules, of various sizes, corresponding to those seen upon the surface. The granules representing the liver-parenchyma are usually opaque, and are separated by the translucent mass of fibrous tissue. The color of the diseased liver is especially modified by the presence of bile-pigment and fat, and then becomes of a tawny-yellow (*κιρρος*) color, whence the much-abused and confusing term "cirrhotic."

The capsule of the liver is thickened and opaque, especially between the projecting nodules, and may be united more or less intimately by fibrous adhesions to the adjoining peritoneum, especially to that of the diaphragm.

The peritoneum in general is apt to be thickened and opaque, at times infiltrated with blood-pigment, and fluid is found in the cavity. The surface is occasionally studded with minute fibrous granules, pseudo-tubercles—a complication which has not infrequently suggested that a tubercular peritonitis is a not infrequent complication of fibrous hepatitis.

The spleen is enlarged and dense, and the stomach and intestines show appearances characteristic of a chronic catarrh.

The contraction of the new-formed fibrous tissue in the liver leads to the obstruction and obliteration of many of the intrahepatic branches of the portal vein and of certain of the bile-ducts. Appearances suggesting a new formation of the latter are very frequently found. The disturbing effects of obstruction to the flow of portal blood through the liver are partly compensated for by a dilatation of the anastomoses between the branches of the portal system of veins and those of the vena cava. This takes place at the junction of the œsophagus and the stomach, along the course of the large intestine at the lower part of the rectum, and in the retroperitoneal plexus of veins in front of the spine. Communicating veins in the suspensory and round ligament become dilated and perhaps reopened (the umbilical vein), and give rise to the rarely seen circle of veins in the abdominal wall radiating from the navel and called the *caput Medusæ.*

The branches of the internal mammary and epigastric veins also become dilated and tortuous, and carry to the heart much of the portal blood which is unable to pass through the liver.

Symptomatology.—There are no characteristic symptoms which announce the early stages of fibrous hepatitis. Those which are present, as epigastric weight and tension, sensitiveness in the region of the liver, temporary and slight elevations of temperature, are likely to owe their origin to the action on the stomach and intestines of the most frequent cause of the hepatitis—viz. alcohol.

In general the symptoms are those due to the obstructed portal circulation, and eventually to the destruction of the liver-cells. Among the former are to be included the loss of appetite, belching, nausea, vomiting, flatulence, irregular action of the bowels, which are indications of the chronic gastric and intestinal catarrh. Jaundice is usually an infrequent symptom in the ordinary varieties of fibrous hepatitis, and, if present, is but slight. When found it rarely persists for any length of time, but may recur, perhaps, with slight febrile disturbance. Sooner or later hæmorrhages from the digestive canal are likely to occur. The blood is vomited when the bleeding takes place from the distended veins in the stomach or œsophagus, or escapes from the rectum when the bleeding occurs in the intestine. In the latter case it is usually in the small intestine. Large quantities of blood may thus escape, and the hæmorrhages may occur at intervals of weeks or months, extending over a period of a year or more.

Epistaxis and bleeding from piles may take place, although hæmorrhoids, whether blind or bleeding, are less often present than is popularly thought to be the case. Cutaneous hæmorrhages may also occur, although they are apt to take place late in the course of the disease.

The gastro-intestinal hæmorrhages are, at times, the first symptoms suggestive of the existence of fibrous hepatitis, and may give temporary relief to the digestive disturbances. In other cases they do not appear until the abdomen becomes distended with serous fluid.

Ascites is eventually the most conspicuous symptom of fibrous hepatitis, and may be the first to urge the patient to seek medical advice. It is usually slow in development, and is due chiefly to the mechanical obstruction to the passage of portal blood through the liver, and in part to the general disturbance of nutrition which occurs in the course of the hepatitis. It may rapidly develop in the case of a complicating thrombosis of the portal vein.

The quantity of fluid which may accumulate in the peritoneal cavity is sometimes as much as twenty quarts, and the abdominal distention arising from its presence, and the frequent excessive meteorism, may be enormous. The intra-abdominal pressure may vary considerably, even on the same day, and it is evident that temporary variations may take place in the quantity of ascitic fluid, and are especially seen to follow the occurrence of hæmorrhages and the action of salines. Eventually, the accumulation becomes so large that tapping becomes necessary. The fluid reaccumulates in the course of a few weeks, and the ascites thus usually represents a persistent symptom after its first appearance.

Œdema of the legs and feet, of the external genitals, and of dependent portions of the abdominal walls is frequent in the later stages of the disease, and depends in part upon the pressure of the abdominal contents upon the inferior cava and iliac veins, and in part upon the enfeeblement of the general circulation. To the latter cause may be attributed the hydrothorax and œdema of the lungs which are often present toward the close of life.

The heart's action becomes enfeebled, the respiration labored and accelerated, in part from the elevation of the diaphragm in consequence of the abdominal distension.

Fever is not a symptom of fibrous hepatitis. If it be present, it is rather to be attributed to some inflammatory complication of the lungs, pleura, or peritoneum.

With the progressive destruction of liver-cells and the resulting disturbance in the functions of the liver in secretion and in the metamorphosis of tissue, the digestive disturbances become more extreme. The loss of flesh and strength becomes increased and progressive, the patient becomes nervous and irritable, and at the end of life stupor or delirium, coma or convulsions, may be present.

On physical examination there may be a faint, and perhaps recurrent, yellowish tint of the skin, especially in the earlier stages of the disease. Later, the skin has rather an ashen or earthy look, and becomes dry and scaly. The loss of fat-tissue is conspicuous; the eyes are sunken and the cheek-bones prominent. The tongue is coated, rather dry than moist.

The distended abdomen contrasts sharply with the emaciated chest. The navel protrudes, and the superficial veins are prominent. The more considerable the distention, the less possible the satisfactory exploration of the liver and spleen until after the removal of the fluid: the liver then may be found moderately enlarged (in the early stages or when combined with fatty infiltration). More characteristic of the ascitic stage of fibrous hepatitis is the small, contracted liver, the dense edge of which may be felt in the epigastrium and just behind the costal cartilage. The granules and lobules of the anterior surface, when present, are also frequently to be recognized. Percussion is of less value than palpation in determining the condition of the liver, owing to the descent of the lungs and the interposition of the distended intestine.

Enlargement of the spleen is the usual accompaniment of fibrous hepatitis, and is the more extreme the more severe and prolonged the chief cause, the obstructed portal circulation. It may be partly due to the cause of the hepatitis, as in infective cases, especially in malaria, in which case it is of early occurrence. Usually it is one of the later phenomena, and is the smaller the more efficient the collateral circulation or the more profuse the gastro-intestinal hæmorrhages. A thickened capsule or senile atrophy may prevent any considerable enlargement: usually it is doubled or tripled in size, increased in density, and its edge is readily felt, even without prolonged inspiration, below the left costal cartilages. An enlarged area of dulness may be found on percussion, although the same difficulty may be experienced from this method of exploration, as in the attempts to define the outlines of the liver by percussion.

Course and Duration.—Not only are there no characteristic symptoms to announce the onset of a fibrous hepatitis, but the beginnings of the disease are as obscure as the course is latent until the gastro-intestinal hæmorrhages or the ascites suggest its nature. After the diagnosis is made clear the course is usually rapid, and the end reached within a few months or a year. It is possible that the course may be protracted over a longer period if the diagnosis be made early, the causes avoided, and the treatment initiated before the alterations have become considerable. Death usually results from exhaustion or from some acute complication, as a pleurisy, pneumonia, erysipelas, or peritonitis (perhaps following abdominal puncture), or from a degeneration of the heart or kidneys.

Diagnosis.—A drunkard or free liver, who has long suffered from the symptoms of chronic gastric catarrh, and who has had attacks of hæmorrhage from the stomach and bowels, or who becomes gradually ascitic, may well be suspected of having a fibrous liver. This diagnosis receives confirmation from the recognition of an indurated, perhaps granular, liver and a considerably enlarged spleen, especially after the abdominal fluid has been removed by tapping.

In the earlier stages the diagnosis is not to be made, but the habits of the dyspeptic patient serve as a warning. In the later stages of the disease the differential diagnosis between a chronic, a tubercular, or a cancerous peritonitis may be made with difficulty, and indeed may be impossible except by an exploratory laparotomy.

In chronic and in tubercular peritonitis there is more likely to be a febrile elevation of temperature; there is no especial enlargement of the spleen; there may be evidence of a tuberculosis elsewhere. Jaundice is absent, and the skin is less earthy in color. Cancerous peritonitis may also be differentiated with difficulty in the absence of evidence of cancer elsewhere. As a rule, such evidence is present after tapping if not before this operation.

Prognosis.—Fibrous hepatitis, when sufficiently far advanced for the diagnosis to be satisfactorily made, is a progressive and incurable disease. In the early stages, when the diagnosis is in doubt, the possibility of an arrest of the process by the removal of the cause is possible. Even when hæmorrhages and ascites have occurred and patients have been tapped, these symptoms have ceased and patients have lived for years. It is no exceptional experience for considerable degrees of fibrous atrophy of the liver to be found unexpectedly at post-mortem examinations. It is therefore necessary to make a qualified prognosis in the individual case only after a careful consideration of the degree of exposure to the cause, the duration and severity of the symptoms, the apparent size of the liver, and the ability of the patient to remain well-nourished. As a rule, a fatal termination may be expected within a year after the occurrence of gastro-intestinal hæmorrhages and dropsy, the signs of an obstructed portal circulation.

Treatment.—The treatment consists, first, in the removal of all irritating articles of food, especially the avoidance of alcohol, spices, and coffee and in the removal of a malarial patient to a non-malarial climate. The syphilitic patient may be treated with iodide of potash and with mercury, with a doubt as to the success of these remedies in arresting the progress of the hepatitis.

The diet should be largely of milk. Green vegetables and fruit (avoiding potatoes), beans, peas, eggs, boiled fish, and fresh lean meat may be eaten if well borne. Stale bread, especially Graham bread, may be taken. The patient should avoid fatigue and be carefully protected from cold and wet.

The treatment is to be particularly directed to the relief of the gastrointestinal catarrh, hæmorrhages, ascites, and progressive weakness, and will be most efficient in case of the ascites.

Gentian and quassia before meals, hydrochloric acid afterward, chloroform-water for nausea, peppermint-water or ginger tea to relieve flatulence, are all to be recommended.

The action of the bowels is to be regulated by mild doses of salines or by rhubarb, aloes, or calomel. Except in the later stages of the disease or when hæmorrhage is taking place, diarrhœa, if not too profuse, need not be checked by medicine.

Hæmorrhage from the stomach or bowels is most efficiently relieved by ice and morphine. It rarely proves immediately fatal, and there are no lesions of surface to be affected by medicine. Ergot may be given, though it is of doubtful value.

Ascites is the condition for which effective treatment is eventually demanded, and the means to be employed for its relief are described in detail in the article on Ascites.

Toward the end of life alcohol may be necessary as a stimulant, and its use should be advised without question.

HYPERTROPHIC CIRRHOSIS.

An exceptional variety of fibrous hepatitis is that to which the term "hypertrophic cirrhosis" is applied. The condition is so well marked clinically that it deserves an independent position in a work on the practice of medicine.

Persistent enlargement of the liver for years—seven in one instance—is the anatomical condition. There are no histological characteristics by means of which this variety is to be differentiated from the atrophic form.

The most important symptom is jaundice, which is permanent. It may be extreme, yet the fæces be stained with bile. It is this persistent jaundice in hepatitis, with open bile-ducts, which has suggested that chronic inflammation of the bile-ducts may be the cause of hypertrophic cirrhosis.

The digestion is somewhat disturbed, but for a long time the nutrition of the body is unaffected. Eventually, emaciation and debility occur, or the jaundice may rapidly become more extreme, the temperature rise, perhaps to 108° F., delirium and convulsions take place and death speedily ensue.

The absence of ascites and the infrequency of hæmorrhages are conspicuous in this variety of fibrous hepatitis.

The lower edge of the liver may reach to and below the navel. The liver is not tender to the touch, the surface is smooth, and the density increased. The spleen is enlarged.

The **diagnosis** is based upon the presence of persistent jaundice and the evidence of enlargement of the liver and spleen and the absence of ascites and gastro-intestinal hæmorrhages.

An amyloid liver is differentiated by the presence of jaundice, the absence of etiological factors, diarrhœa, albuminuria, and dropsy. The progress of cancer of the liver is more rapid, and this affection is not associated with an enlargement of the spleen. The progress is slow, the condition incurable, and the treatment the same as that prescribed for the atrophic variety of fibrous hepatitis.

FATTY LIVER; FATTY INFILTRATION OF THE LIVER.

The accumulation of fat in the liver is called fatty infiltration or fatty degeneration of the liver, according as the fat-drops are considered to lie in an otherwise normal cell or in one which has lost a certain amount of its albuminoid constituents. In the former instance the fat is supposed to be derived from some constituent of the food, notably from fat or from carbohydrates; in the latter from metamorphosis of protoplasm. This distinction is rather conventional than actual, since the emaciating individual may have an accumulation of fat in the liver derived from the metamorphosis of cell-protoplasm in some other part of the body than in the liver.

For practical purposes, the fatty liver is to be regarded as altered in virtue of accumulated fat, in contradistinction to one in which fat is accumulated as the result of a degeneration of cell-protoplasm. The latter further represents a condition analogous to the alteration to be found in acute yellow atrophy of this organ, acute parenchymatous hepatitis, and is found in phosphorus-poisoning, the acute fatty degeneration of the new-born, and of pregnancy.

Etiology.—The liver is generally regarded as one of the organs in which fat derived from the fat and carbohydrates of the food is normally accumulated. Hence a certain degree of fatty infiltration is to be considered as a normal condition of this organ. The fat may remain accumulated for a considerable time, as in the fatty infiltrated livers of infants, or it may be of brief and periodical occurrence, as takes place after meals in which a fatty or fat-forming diet forms an important part.

Since fat is considered to be removed from the liver by its oxidation in the blood and by its elimination through the bile, processes which check oxidation and increase the secretion of bile favor the retention of fat in the liver, and active oxidation represents a most efficient cause of the increased removal of fat from the biliary lobules.

The immediate causes of a fatty liver are thus to be found in large eaters, especially those partaking freely of abundant fatty, saccharine, and starchy food. Since such individuals usually take insufficient exercise, the fat absorbed from the food is likely to be incompletely oxidized; thus an additional element in its retention is present.

Chronic alcoholism is another important factor in the etiology of fatty liver. Deficient oxidation is a conspicuous feature in such cases.

Extreme anæmia, whether from repeated hæmorrhages or from unknown causes, as in progressive pernicious anæmia, is accompanied by a fatty liver, largely in virtue of defective oxidation from scanty red blood-corpuscles.

Wasting diseases, especially pulmonary consumption, produce a fatty liver, partly from the destruction of lung-tissue and the associated anæmia producing deficient oxidation, partly in virtue of the emaciation, representing an excessive albuminoid metamorphosis at remote parts of the body, and partly in consequence of the abundant fatty diet (cod-liver oil, milk, and cream) usually employed as a therapeutic measure in such diseases.

Chronic diarrhœa is also an emaciating disease, in which a fatty liver owes its origin to some of the causes concerned in producing the fatty liver of pulmonary phthisis.

Rickets and malaria are affections in which a fatty liver is more rarely met with, and then, probably, in virtue of the same disturbances of nutrition and oxidation as those already considered.

Morbid Anatomy.—The more extreme degrees of fatty liver are characterized by an increase in size and weight, the liver often being doubled in size. The enlargement is symmetrical, the edges are rounded, and the consistency is notably diminished, the surface pitting on pressure. The color is pale yellow, and clusters of injected veins are frequently seen beneath the smooth and shining capsule.

On section the lobules are large, often indistinctly defined, and there is a conspicuous ischæmia of the organ. The knife used in cutting is besmeared with fat. The gall-blader often contains a thin, pale-yellow, watery bile.

Symptomatology.—In the multitude of symptoms which are present in the conditions with which a fatty liver is associated there are none which are absolutely characteristic of this condition. It is only the more extreme degrees of fatty liver which may be considered to produce a direct disturbance, especially in virtue of the large size attained by this gland. A sense of fulness and tension, with perhaps a slight degree of pain in the hepatic region, may be complained of. Digestive disturbances are also frequent, usually attributed to obstruction to the portal circulation, resulting from the compression of the portal capillaries in the liver by surrounding fat. These are loss of appetite, retching, nausea, vomiting, constipation or diarrhœa. The stools may be pale and offensive, conditions attributed to a lack of bile in the intestines. The portal obstruction is never sufficient to produce enlargement of the spleen or ascites, and the deficiency of secretion or its obstructed outflow is insufficient to produce jaundice. There is no febrile disturbance, and the urine in few cases of fatty infiltration is altered in quantity and quality.

The enlarged liver may reach to the navel, although its outlines in a fat person are determined with difficulty, in consequence of the abundant subcutaneous and omental fat. There is but little resistance to the palpating finger for the same reason, and also from the fact that during life the resistance of the fatty liver is markedly diminished.

Course and Duration.—The fatty liver is a chronic affection, and progresses but slowly. If the cause be remedied, a return to the normal is possible; if a fatal disease be the cause, the fatty liver persists to the end of life.

Diagnosis.—A fatty liver is to be recognized only when it has attained a considerable size. An enlarged liver being determined by physical examination, its fatty nature may be inferred from the presence of one of the exciting causes. It is to be differentiated from the large fibrous and amyloid liver in virtue of a lesser density and definition and by the lack of the persistent jaundice, enlarged spleen or ascites to be found in fibrous hepatitis, and by the absence of the cachexia, œdema, and albuminuria connected with the amyloid liver. The hyperplastic liver of leukæmia is to be differentiated by the abscence of increase of leucocytes in the blood.

In pulmonary phthisis an enlarged liver may be either fatty or amyloid, but the indications above mentioned will usually suffice for the differential diagnosis, although the case may arise where this becomes impossible.

Prognosis.—Recovery from a fatty liver is possible when the causes are transitory, and the affection cannot be regarded as a cause of death or as a serious complication of the disease in which it may occur.

Treatment.—Since an excessive fat-forming diet and deficient oxidation favor fatty infiltration of the liver, the treatment consists, in the first instance, in the careful, not too extreme and sudden, regulation of the diet and exercise. The use of fat, sugar, and starch is to be restricted, and the patient is to be advised to live largely on lean meat, fish, green vegetables, and fruit. The phthisical patient should not be deprived of his cod-liver oil, milk, butter, and cream, since experience shows that the general condition improves under the abundant use of these articles of food.

Exercise should be freely taken up to the point of fatigue.

The digestive disturbances are to be controlled by the use of bitters, such as teaspoonful doses of the compound tincture of gentian, of the tincture or elixir of iron, quinine and strychnine, or of the compound tincture of cinchona.

Constipation is to be overcome by the use of rhubarb, aloes, or colocynth, and diarrhœa is to be controlled by the administration of opium.

Intestinal fermentation may be checked by pills of ox-gall or by the use of equal parts of resorcin, bismuth, benzo-naphthol, and aromatic powder, a teaspoonful every two hours, as recommended by Ewald.

In the anæmic form of fatty liver a preparation of iron is to be advised.

AMYLOID LIVER.

Amyloid degeneration, or infiltration of the liver, is the term applied to the presence of amyloid material in the blood-vessels and interstitial tissue of the liver.

Etiology.—The method of origin of amyloid material is unknown. Its presence in the liver occurs in the course of certain affections, the only known factors in etiology. The most important of these affections are chronic suppurative processes, especially those of a tuberculous nature, in the bones and joints. Chronic tuberculosis of the lungs and kidneys, especially when connected with extensive ulceration, is of like significance. Extensive chronic ulcers of the skin and intestines are an occasional antecedent. More important is syphilis, especially in the later stages. Amyloid livers are sometimes found in cancerous affections, in rickets, malaria, gout, leukæmia, and pseudoleukæmia, and in chronic nephritis.

Morbid Anatomy.—Since amyloid degeneration of the liver is a progressive affection, various degrees of alteration may be found. In the extreme cases the liver is greatly and symmetrically enlarged, more than doubled in size, and increased more than threefold in weight. The color is of a pale reddish-gray, and the consistency greatly increased. The capsule is tense, smooth, and shining, and the section shows a pale-gray, homogeneous, translucent surface in which streaks and spots, often of an opaque yellow color, are indicative of those portions of the liver less infiltrated with the amyloid substance. The translucency of the degenerated liver is conspicuous when the light is transmitted through thin slices; the translucent amyloid portions are stained of a mahogany-brown color when the surface is covered with a compound solution of iodine.

The gall-bladder is apt to contain a small quantity of pale, watery bile.

Symptomatology.—There are no characteristic symptoms of amyloid degeneration of the liver. The patient is pale, cachectic, and, in the later stages of the disease, œdematous and dropsical. These conditions are not directly attributable to the amyloid affection of the liver, since when this organ is degenerated the same condition is likely to be found in the spleen kidneys, or intestines. A considerable enlargement of the liver may give rise to a sense of fulness and weight in the right hypochondrium.

Although there is an extensive destruction of liver-cells and a marked obstruction to the flow of portal blood through the liver, it is doubtful to what extent the disturbances of digestion, if present, are due to a deficiency of bile, as it is doubtful whether any ascitic fluid present is to be regarded as the result of a mechanical dropsy. The digestive disturbances are loss of appetite, gaseous eructations, vomiting, and diarrhœa. The stools may be pale, but there is no jaundice. The diarrhœa is often attributable to the associated amyloid degeneration of the intestines. There is no fever. The urine is usually of low specific gravity, pale, highly albuminous in virtue of the associated amyloid affection of the kidneys.

The physical examination of the liver indicates the degree of enlargement. The dulness may extend from the third rib to the level of the crest of the ilium. On palpation the enlarged liver is smooth and tense, its edge sharply defined. It is neither painful nor tender.

The spleen may or may not be enlarged. In the latter case the increase in size is rather due to an amyloid infiltration of the organ than to its passive congestion.

Course and Duration.—Amyloid degeneration of the liver is usually a chronic affection, progressing gradually, increasing in extent during a period of months or years, although the diagnosis is usually made within a few months of death. Its progress is sometimes very rapid. Cohnheim has reported a case of extreme amyloid disease which had developed in the course of three months.

Diagnosis.—The diagnosis of an amyloid liver is based upon the occurrence of enlargement of this organ in a patient with one of the above-mentioned antecedents, who is anæmic, cachectic, and dropsical. The diagnosis is supported by evident enlargement of the spleen and by the presence of diarrhœa and albuminuria.

Prognosis.—It is doubtful whether a patient with amyloid liver ever recovers from the disease in which it occurs. Although it is possible that slight degrees of amyloid degeneration may remain stationary, and perhaps be recovered from after removal of the cause, the patient usually becomes paler, more emaciated, and eventually dies dropsical and exhausted.

Treatment.—Since the recognition of an amyloid liver usually precedes death by a few months only, there is no treatment for this affection. Its occurrence is only to be prevented by a removal of the cause.

The efficient early surgical treatment of the diseased bones and joints, and the effective medical treatment of the various lesions of syphilis, and the arrest of the progress of tuberculosis of the lungs and intestines, may prevent the spread of amyloid disease. In general both in the liver and elsewhere, it represents a terminal stage of the fatal disease in which it occurs. The symptoms connected with its presence are to be treated as they arise, and demand nourishing diet, tonics, laxatives, and astringents.

Cancer of the Liver.

Tumors of the liver are to be distinguished for clinical purposes into those which are *benign* and those which are *malignant.* The former group includes myxoma, fibroma, lipoma, glioma, angioma, retention-cysts, and a part of the adenomata, while the malignant tumors are represented by certain adenomata, sarcoma, and cancer. For convenience the term "cancer" will be applied to the latter series, the different members of which, under circumstances, usually require a microscopical examination for their exact differentiation. The *malignant tumor* of the liver is usually a cancer or a sarcoma, and has been found in about 3 per cent. of a large number of cases of death from various causes.

Etiology.—Cancer of the liver is usually secondary to cancerous disease elsewhere, more commonly in those organs containing the radicles of the portal vein. Hence it is likely to follow cancer of the alimentary canal, especially of the stomach, rectum, colon, and œsophagus; cancer of the gall-bladder or common duct or of the pancreas, may be the primary affection. It may follow cancer of the uterus, ovaries, or mammary gland. Sarcoma of the liver is usually secondary to sarcoma of the skin, eye, brain, and membranes or bones. Both cancer and sarcoma may arise primarily in the liver, but they are usually secondary to growths in other parts.

Cancer of the liver occurs, as a rule, after middle life, though it has been found in infants and young children. It is more common in women than in men, presumably in consequence of the frequent primary occurrence of cancer in the sexual organs of the female. Heredity, injury, and gall-stones are assigned a certain degree of importance in etiology. Although gall-stones are not infrequently found in cancer of the liver, they may be a result, as well as a cause, of this affection.

Morbid Anatomy.—Malignant disease of the liver occurs in the form of nodules or as a diffuse infiltration of the organ. Either variety may be primary or secondary, and its growth is associated with absorption of the liver-cells. The nodules may be single or multiple, the latter being the usual characteristic of secondary growths. The right lobe is apt to be more especially diseased, although, exceptionally, the left lobe may be predominantly affected.

As a rule, the liver is increased in size, sometimes enormously, then weighing as much as twenty-five pounds. The increase in size is symmetrical when the organ is infiltrated; when nodules are present the enlargement may be extremely irregular. The nodules vary in size from that of a pinhead or less to that of the fist or upward. They may occur in great numbers, more than a hundred. Some of the nodules project from the surface of the liver, either rounded or flattened, the latter often presenting a central depression, the umbilicated appearance. Smaller nodules are often to be found at the periphery of the larger nodules, the latter appearing in considerable part to arise from the confluence of the smaller nodules. The peritoneum is often thickened and opaque, and may contain dilated and tortuous blood-vessels.

On section of the liver the nodules present a globular shape, and when small are of a gray, reddish-gray, or white color, though some are dirty gray or black (melanotic sarcoma or cancer). As the nodules become larger they assume a variegated appearance in consequence of secondary changes. Fatty degeneration and necrosis of the cells give a yellow color or a caseous look. Hæmorrhages produce shades of red and brown corresponding to the extent and age of the bleeding. The centre of the nodule may be transformed into a cyst-like cavity containing a thin, opaque, yellow fluid.

The consistency of the nodules varies extremely, and the medullary or encephaloid, the fibrous or scirrhous, the hyaline or gelatinous appearances are to be observed.

The infiltrating cancer of the liver produces a more symmetrical enlargement of the liver, which thus attains its greatest size. The enlargement is more or less, not absolutely, symmetrical, and in extreme instances the vertical diameter shows the greatest increase. The color is apt to be paler than normal unless the growth be melanotic, in which case the appearance of the section of the liver has been compared to that of granite. The infiltration may be so symmetrical that the structural details of the normal liver are closely followed, or interlacing bands of dense fibrous tissue may be formed enclosing islets of cancer, presenting an appearance simulating that of fibrous hepatitis.

Both in the nodular and infiltrating varieties the cancer may compress and penetrate branches of the hepatic and portal veins, narrowing and obliterating their canals, with or without the formation of thrombi. Corresponding disturbances in the circulation of the affected parts of the liver result, and are manifested by injection, infarction, and necrosis. Compression of the trunk of the portal vein may occur from the extension of the cancer to the lymph-glands in the portal fissure, and perforation of its walls or that of the gall-bladder may result from extension of the disease in the liver. In like manner, pressure upon the bile-ducts gives rise to dilatation and jaundice.

Cancer of the liver is often associated with peritoneal cancer, and the lungs, spleen, or kidneys may be similarly affected.

Symptomatology.—The disease in the liver may be so little advanced as to give rise to few or no symptoms. The more extensive the cancer of the liver, the greater the resulting disturbances, although for a time they may yield in importance to the symptoms due to the primary cancer. Digestive disturbances are usually present. Loss of appetite, nausea, vomiting, and constipation become associated with emaciation and debility.

Pain in the hepatic region is almost constant, and radiates outward in various directions. In at least half of the cases jaundice is present, with or without colored stools. It becomes persistent, and sometimes extreme. In a like number of cases the abdomen becomes distended with fluid, either ascitic from pressure upon or obliteration of the portal vein, or peritonitic from simple or cancerous peritonitis.

Cutaneous hæmorrhages or bleeding from the nose, stomach, and bowels may occur late in the disease, and itching of the skin and hiccough are sometimes troublesome. The patient becomes cachectic, and the skin may show a certain degree of œdema. The pulse is slow when jaundice is present, and the temperature is often normal, perhaps subnormal toward the end of life. On the contrary, a febrile temperature is sometimes present, especially when the disease runs a rapid course or is accompanied with suppuration. The fever may be continuous or intermittent.

The urine is diminished in quantity, high-colored, even in the absence of jaundice, and may contain a trace of albumin. The indican is increased, and in melanotic cancer or sarcoma melanin or melanogen may be present. The urine then may be dark, almost black, when passed, from the presence of melanin, or it may become black after standing or when heated with nitric acid, from the presence of melanogen.

The liver is usually tender, and may be so enlarged that the area of hepatic dulness extends from the third right rib to below the iliac crest. In nodular cancer the nodules are to be felt, and sometimes seen, through the thin abdominal wall, and, if the liver be not too large, move with the diaphragm. They vary in consistency, and large nodules may sometimes be so soft as to suggest the presence of fluid. In such cases an exploratory puncture is likely to remove a quantity of blood which offers little or no evidence as to its source. If the left lobe be especially enlarged, it may transmit the aortic impulse so strongly as to suggest an aneurism of this vessel. The pulsation, however, is not expansile. As in case of the aneurism, the spleen is usually not enlarged.

Course and Duration.—The progress of the disease is in the direction of increasing loss of flesh and strength, and may be so rapid that death occurs in the course of a few weeks. The average duration of the disease after its recognition is some four or five months, although it may extend over a period of more than a year. Death may result from the progressive debility with eventual pulmonary œdema, or it may be due to cancerous peritonitis or to intraperitoneal hæmorrhage from rupture of a nodule.

Diagnosis.—The concurrence of a progressive loss of flesh and strength with pain and tenderness in the right hypochondrium, and an enlarged nodular liver, would lead to a diagnosis of cancer of the liver. This diagnosis would be strengthened by the presence of jaundice and ascites and nodules connected with the peritoneum. An enlarged nodular liver might be produced by an echinococcus cyst, but the latter increases in size without much constitutional disturbance, and an exploration would make the diagnosis clear. Nodular deformity of the liver from fibrous hepatitis would lack the cachexia of cancer.

With the above symptoms and a symmetrically enlarged liver the question of a hypertrophic cirrhosis might be raised. In the latter affection the cachexia is of late occurrence and the spleen is enlarged. In amyloid liver, the antecedents are pronounced, the disease is of slower progress, jaundice is infrequent, albuminuria and dropsy are greater.

Greater difficulty in diagnosis is likely to arise in discriminating between cancer of neighboring, perhaps adherent, organs and cancer of the liver, or in recognizing cancer of the liver without any notable enlargement of this organ. Under such circumstances the differential diagnosis might become impossible.

Treatment.—Malignant disease of the liver is inevitably fatal, and its treatment consists merely in the relief of symptoms, especially in the use of laxatives and opiates.

PARASITES OF THE LIVER.

Of the parasites which may be found in the liver, there are three which have little or no clinical importance. These are the pentastomum denticulatum, the cysticercus cellulosæ, and the psorosperms. They are extremely rare, and produce little or no disturbance. The echinococcus, on the contrary, is often met with in some countries, is productive of serious if not fatal results, and its recognition is of increasing importance from the success which has attended efforts at its removal.

ECHINOCOCCUS; HYDATID OF THE LIVER.

Etiology.—The echinococcus is the cestode or larval stage of a tape-worm, the *Tænia echinococcus.* The latter is found in the small intestine of the dog or of allied animals, the wolf and jackal. It is exceedingly minute, 4–5 millimetres long, and easily escapes recognition. It is composed of a head surmounted by a rostellum with a double row of hooklets, twenty or thirty, and four suckers. To the head are attached four joints, only the last being mature and bearing eggs capable of producing its like. The eggs, when swallowed by man, are believed to perforate the intestinal wall and enter the liver by means of the portal vessels. Within the liver they are transformed into the echinococcus or hydatid cyst.

Since man is usually infected by swallowing the ova from the tænia of the dog, it follows that this event is more likely to occur the more intimate the relation between man and dog. It is, consequently, in such countries as Iceland and Australia that this hydatid disease is most often found, although it is not unusual in other civilized countries. In the United States and Canada it is comparatively rare, according to Osler, who has been enabled to collect only 85 cases. It is more often found among the poor and among those whose occupation—as shepherds, for instance—renders them especially liable from their uncleanly habits and intimate relation to the dog.

Morbid Anatomy.—After the embryo has reached the liver it continues to increase in size and becomes transformed into a cyst. Either a single cyst is found, the unilocular echinococcus, or innumerable cysts are present, the multilocular echinococcus.

The unilocular echinococcus is the variety most often found. It may be present in either lobe of the liver, usually the right, and may be seated near the surface or deep in the organ. It may increase in size until it produces an enormous enlargement of the liver, extending from the second rib to the crest of the ilium, or it may project above the surface as a circumscribed tumor, sometimes pedunculated. When the echinococcus is superficial, the fibrous peritoneal covering of the liver is likely to become thickened and opaque, and adhesions may form between it and neighboring parts, whether visceral or parietal. The parasites enlarge by the accumulation of fluid within the parent cyst or by the formation of daughter and granddaughter cysts, until thousands may be present. The parent cyst has a smooth, homogeneous, opaque, lamellated wall, readily becoming convoluted after removal of its contents. The inner surface of this wall may be studded with brood-capsules and scolices. The latter are the immature tapeworms, and project from or are inverted into the capsules. They have a head, rostellum, suckers, and hooklets, like the ancestor. Some of these cysts may be sterile, having no scolices. The parent cyst and its offspring may contain a large or small quantity of clear, pale-yellow liquid, the specific gravity of which varies between 1005 and 1015. It is free from albumin, contains abundant chlorides, a trace of sugar, and succinic acid. The presence of the latter is determined by its changing to a brown color on the addition of a dilute solution of ferric chloride. The parent cyst lies in a fibrous capsule developed from the fibrous tissue of the liver, and its inner surface may be stained of an iron-rust color from the presence of cystalline hæmatoidin or bilirubin.

The liver-tissue becomes atrophied in proportion to the number and size of the cysts, but a regeneration of liver-tissue has sometimes been observed.

The echinococcus may die, as from injury or the action of bile, in which case a part of its fluid becomes absorbed, the cyst-wall becomes convoluted and contains a yellow greasy material in which fat-drops, crystals of cholesterin and hooklets may be found. The fibrous capsule may become inflamed, and an abscess arise, perhaps resulting in the death of the echinococcus. Lime salts may become deposited in the remains of the dead echinococcus, from which the hooklets may be removed after solution of the salts.

The multilocular echinococcus forms a relatively solid and somewhat irregular tumor intimately connected with the liver, and may attain a great size. It has a honeycombed structure, being traversed by fibrous septa enclosing more or less rounded spaces in which is a liquid or gelatinous material, the daughter cyst. These are usually sterile, but may contain scolices and hooklets.

The peculiar characteristics of the multilocular echinococcus are attributed to a growth of the brood-capsules into pre-existing spaces, lymphatics, blood-vessels, or bile-ducts, from the outside wall of the parent echinococcus.

If echinococci are found in the liver, they are not infrequently found elsewhere, as in the peritoneum and spleen, in the heart, lungs, and brain.

Symptomatology.—Small echinococci produce no symptoms; large cysts or those favorably situated may give rise to serious, even fatal, results by pressure, rupture, or suppuration. Usually there is but little disturbance of the nutrition of the patient.

In case of extreme enlargement of the liver the diaphragm may be pressed upward, the heart and lungs displaced, and severe dyspnœa result. The portal vein may become compressed and ascites follow, or the hepatic vein or vena cava may become obstructed and œdema of the legs take place. If the main bile-ducts are obstructed, jaundice will follow. The pressure of the enlarged liver in the right hypochondrium may give rise to a sense of weight and distention, and may produce a bulging of that region. Pressure upon the sensory nerves supplying the legs may result in pain, numbness, or prickling.

Rupture of the cyst may take place into the peritoneum, pericardium, or pleura, with a resulting acute, perhaps fatal, inflammation of these membranes. Urticaria has repeatedly been observed to follow rupture into the peritoneal cavity. The rupture may take place into the lung, and pneumonia follow, or cysts, scolices, or hooklets may be coughed out of the lung for a period of months. More frequently pus, blood, bile- or blood-pigment may be raised, and the patient appear to be suffering from an abscess or gangrene of the lung. The perforation may take place into the gall-bladder, with symptoms of gall-stone. If into the stomach or intestine, cysts may be vomited or escape through the bowels. Rupture into the pelvis of the kidney has been observed also through the abdominal wall, with the discharge of the hydatids. Fatal embolism has resulted from rupture into the hepatic vein and the passage of hydatids into the heart and pulmonary artery.

Suppuration of the fibrous capsule of the cysts results in the formation of an abscess, which may lie within the liver or assume the characteristics of a subphrenic abscess. Chills, fever, jaundice, and progressive emaciation may result from the complication. The abscess may break, as may the cyst itself, into neighboring parts.

The nature of the enlargement of the liver is to be determined, in the main, by the results of the physical examination. Inspection may reveal a deformity of the epigastrium or right hypochondrium. Percussion will show an increased area of dulness in the region of the liver conforming to the size of the cyst. If this project into the thorax from the upper surface of the liver, it is important to determine its outlines, which are likely to be curved upward, the uppermost point being in the axillary region, whereas in pleuritic exudation the highest point is in the dorsal region. By palpation nodules may be felt moving with the diaphragm when motion is possible. The nodules are elastic, perhaps fluctuating, and may present the hydatid thrill, compared to the quivering of jelly, although this sign is not to be regarded as pathognomonic of this disease.

The multilocular echinococcus appears rather as an irregular, dense enlargement of the liver. It is likely to be associated with an enlarged spleen, gastro-intestinal hæmorrhages, ascites, and jaundice.

Diagnosis.—The diagnosis of echinococcus of the liver rests essentially upon the recognition of an enlargement or tumor of the liver and the determination of its contents, especially in the discovery of hooklets, by means of the aspirator. Enlargement of the liver from fibrous hepatitis is eliminated by the absence of the usual antecedents, ascites and enlarged spleen. Amyloid liver is also to be excluded by the lack of the usual forerunners, by the density of the symmetrically enlarged organ, and by the evidence of like disease elsewhere, especially in the kidneys. Abscess of the liver is to be discriminated from simple hydatids by the chills, fever, and rapid emaciation. It may be impossible to differentiate the abscess from the suppurating hydatid. The enlarged liver from cancer is likely to be present in elderly people, in whom there is pronounced cachexia, frequently with jaundice.

Circumscribed hydatids might be mistaken for pleuritic effusion, in which the line of dulness will differ as above stated, and the heart will be pushed outward, not upward. The subphrenic abscess will be characterized by pain and fever, and, in the end, by the results of aspiration. In like manner, the aspirator will serve to eliminate a dilated gall-bladder or a hydronephrosis. A cystic kidney is likely to be manifested by urine presenting the characteristics of a fibrous atrophy of this organ.

Prognosis.—Echinococcus of the liver is a chronic affection, of slow progress, and may exist for thirty years. A spontaneous cure by death of the parasite is not infrequent, and the more serious complication of rupture and discharge of the cysts through a hollow organ or through the abdominal wall may result in recovery. If sufficiently advanced to permit recognition, it must be regarded as a serious affection in virtue of the complications which may arise.

Treatment.—The treatment begins only with the recognition of the disease, and its aim is to destroy or remove the parasite. Various measures have been adopted for this purpose, of which puncture has been most generally employed. Death of the echinococcus has resulted from puncture and aspiration, puncture and drainage, puncture and injection—*e. g.* with tincture of iodine. Of these methods, the first is uncertain ; the others are dangerous as favoring sepsis. At present the treatment is chiefly surgical, consisting in incision of the sac and evacuation of its contents, either directly after suturing the surface to the lips of the wound, or after obtaining adhesions between the opposed peritoneal surfaces by preliminary operation.

Electrolysis has been used successfully in a number of instances, but the surgical treatment is that to be especially commended as radical and not unduly dangerous.

INFLAMMATION OF THE GALL-DUCTS AND GALL-BLADDER (CHOLANGITIS AND CHOLECYSTITIS).

Inflammation of the biliary tract may be confined to the common or hepatic duct, to the cystic duct or to the gall-bladder, or to a limited part of the ducts.

Etiology.—Inflammation of the biliary tract usually results from the extension of an inflammatory process into the common ducts from the duodenum. The persistence and severity of the inflammation largely depend upon the associated presence of some mechanical irritant, especially gall-stones. The duodenal inflammation is usually part of an acute gastro-duodenitis, excited by irritants taken into the stomach as food and drink, medicines and poisons. Inflammation of the biliary tract also occurs in the course of infectious diseases, as pneumonia, typhoid and malarial fevers, erysipelas, or as an epidemic from more or less unknown influences. It may occur in the course of acute and chronic inflammation or cancer of the liver, and be caused by parasites of this organ or of the ducts. Obstruction to the outflow of blood from the liver in chronic valvular disease of the heart and in emphysema of the lungs also acts as a cause.

Morbid Anatomy.—The inflammation may be limited to the common duct, especially to its duodenal end, or be extended along the common and hepatic ducts to the liver. The cystic duct or gall-bladder may alone be affected, or both may be simultaneously inflamed.

The appearances vary according to whether the inflammation is catarrhal or suppurative. In the mildest form of catarrhal inflammation there may be little found to indicate its presence. Both redness and swelling of the mucous membrane are likely to subside after death, and the mucous and cellular exudation may be slight. A catarrhal inflammation of the duodenal end of the common duct is suggested in a case of jaundice by the presence of a mucous plug, or by the absence of a bile-stained mucous membrane at this part, while toward the liver the stain is apparent. When catarrhal inflammation affects the gall-bladder or the ducts beyond the duodenal end, the appearances may be negative or redness and swelling of the mucous membrane may be found and a more or less viscid, opaque-gray cellular exudation be seen on its surface.

If the catarrhal inflammation of the gall-bladder be associated with obstruction of the cystic duct from whatever cause, the bladder becomes dilated into a pear-shaped cyst, which may hold a quart or more of fluid (dropsy of the gall-bladder). Its mucous membrane then becomes thin, smooth, and shining, and the fluid present is thin, pale, watery, or slimy, containing no bile. The walls of the dilated gall-bladder may eventually become thickened and infiltrated with lime salts.

In suppurative inflammation the mucous membrane of the ducts and of the gall-bladder is thickened, injected, ecchymosed. The ducts are dilated, and their cavity, as well as that of the gall-bladder, contains a purulent or muco-purulent fluid, unstained by bile. The intrahepatic branches of the hepatic duct may be filled with pus. The wall of the inflamed duct or gall-bladder may be extensively ulcerated or covered with patches of necrotic tissue or with fibrinous false membrane.

With the extension of the inflammatory process to the surrounding liver-substance multiple small abscesses are formed. The inflammation may extend from the gall-bladder and extrahepatic ducts to the surrounding parts, with or without perforation, and lead to the establishment of fistulæ between the biliary and intestinal, urinary or respiratory tracts, or between the gall-bladder and skin. Fatal peritonitis follows the discharge of the abscess into the peritoneal cavity.

When the cystic duct is obliterated and a suppurative cholecystitis is present, the gall-bladder is dilated and filled with pus—empyema of the gall-bladder.

Although the tendency of suppurative inflammation of the biliary tract is toward necrosis and perforation, the ulcerated surfaces may become united, causing obliteration and dilatation, or form scars, producing various degrees of deformity.

Symptomatology.—Inflammation of the gall-ducts and gall-bladder is first announced by jaundice, which increases in intensity within the course of a few days. Since the inflammation is usually due to an antecedent gastro-duodenal catarrh, the symptoms of the latter affection—viz. loss of appetite, nausea, vomiting, belching, epigastric tenderness, with more or less irregular stools—are likely to have preceded the appearance of the jaundice by a few days or by a week or two.

The temperature is likely to be slightly elevated, and the area of hepatic dulness is somewhat increased, the edge of the liver being tender.

In catarrhal inflammation of the gall-bladder with obstructed outlet jaundice is unlikely to occur, and there are no symptoms except a sense of pressure or sensitiveness in the region of the distended gall-bladder. The latter is determined by the circumscribed resistance, more or less pear-shaped, dull on percussion, apparently intimately connected with the liver, with which it rises and falls in respiration. Eichhorst refers to a case where the pear-shaped tumor was separated from the lower edge of the liver by the overlying intestine.

If the catarrhal inflammation extend into the smallest gall-ducts, it tends to become persistent and to give rise to that variety of fibrous hepatitis known as hypertrophic cirrhosis.

If the inflammation become suppurative, the patient will suffer from chills, recurring at irregular intervals and followed by fever and sweating, the temperature being elevated to 103° F. and upward. The hepatic tenderness becomes marked. These symptoms may persist for months, the patient becoming progressively emaciated and debilitated, with symptoms of septicæmia. Osler refers to a case of cholangitis with gall-stones in which these symptoms extended over three years, with eventual recovery. Usually the suppurative cholangitis or cholecystitis in the course of a few weeks or months results in perforation with its sequelæ, in abscess of the liver, pylephlebitis, or acute endocarditis.

Diagnosis.—The diagnosis of acute catarrhal cholangitis is usually readily made after the jaundice is present in virtue of the history of the patient and the digestive symptoms. It is distinguished from the effects of gall-stones by the absent biliary colic and by the slow progress of the jaundice. The dilated gall-bladder is to be recognized as an abdominal tumor, and may attain such a size as to be confounded with a cystic tumor arising in the pelvis. It is more likely to be mistaken for a movable kidney, which has a different shape, is more readily displaced, and does not move so distinctly with the rise and fall of the diaphragm. Although the distended gall-bladder is elastic and perhaps fluctuant, the pressure of the contents may be so great as to suggest a solid tumor. It may then be confounded with a cancer of the liver or gall-bladder, which is likely to be harder and more nodular and associated with jaundice and cachexia. It may also be mistaken for an echinococcus cyst, which is likely to have a more globular shape and a broader base. The use of the aspirator may be necessary to establish the differential diagnosis.

The diagnosis of a suppurative cholangitis is only to be suspected in the course of time, when the symptoms of peritonitis or pylephlebitis or of abscess become evident.

Prognosis.—Simple catarrhal cholangitis is usually a mild disease, terminating favorably in the course of a few weeks, the jaundice eventually disappearing in the course of three or four weeks. If it remain slight but persistent for months and years, it is likely to be a part of the hypertrophic cirrhosis already mentioned.

A dropsical gall-bladder from catarrhal cholecystitis rarely ruptures, and produces little or no disturbance.

The prognosis of suppurative cholangitis and cholecystitis is always grave, in virtue of the liability of abscess-formation, thrombo-phlebitis, peritonitis, and septicæmia.

Treatment.—The treatment of catarrh of the bile-ducts is essentially the same as that of acute gastric catarrh. The diet should be largely liquid—milk, broth, and soups—followed by diluted muriatic, phosphoric, or nitro-muriatic acid. Carlsbad salts, Vichy water, or phosphate of sodium are to be taken freely for the purpose of promoting evacuation from the bowels.

Mechanical attempts at emptying the gall-bladder by compression and electricity are not to be employed.

Daily rectal enemata of one to two quarts of cold water are claimed by Krull to produce improvement within a few days. The treatment in general is essentially the same as that for Acute Jaundice, under which head it is more fully described.

The dilated gall-bladder may be treated, if necessary, by puncture or by incision.

Suppurative cholangitis is to be treated like the catarrhal variety. Stimulants may become necessary, and surgical treatment be demanded for an actual or threatening peritonitis. Empyema of the gall-bladder, when recognized, is to be treated surgically.

CHOLELITHIASIS.

The term cholelithiasis includes the method of origin, lesions, symptoms, and the results due to the presence of gall-stones.

Etiology.—Gall-stones are formed within the hepatic ducts and in the gall-bladder. Their formation in the cystic duct and in the common duct is possible, but when found in these portions of the biliary tract they have probably come from the gall-bladder or hepatic duct. Their method of origin is uncertain. According to the chemical theory, their chief constituents, cholesterin and pigment-lime, are precipitated from the bile in virtue of the decomposition of the cholate salts, which are capable of dissolving them. Naunyn, however, claims the cholesterin is formed from the degenerated epithelial cells of the biliary tract, and that the stagnation of the bile is one of the most important factors in the formation of the stones. It is also probable that a catarrhal condition of the biliary tract favors the formation of calculi in its course.

Gall-stones are found much more often in women than in men—four times as often, according to Naunyn—and especially in women who have borne children or from whom abdominal tumors have been removed. They are found in the highest proportion in persons over sixty years of age, but may be met in infancy and childhood. They usually produce disturbances in people from thirty to fifty years of age. Sedentary habits and tight lacing are considered as favoring the formation of the calculi by interfering with the flow of bile. A similar result is attributed to a movable liver or right kidney. Fat people are observed to be rather more frequently affected than the lean. In rare instances foreign bodies, as seeds, parasites, mercury, a needle, and clotted blood, have been found in the centre of a gall-stone.

Morbid Anatomy.—Gall-stones are either homogeneous or nucleated, and are composed of cholesterin alone or of bile-pigment and lime, usually bilirubin-lime either alone or combined with cholesterin; calcic carbonate may be present to a certain extent, and rarely traces of iron or copper. By treating portions of a dried calculus with alcoholized ether the cholesterin is dissolved. If the solution be evaporated on a watch-glass, crystals of cholesterin will be found. The presence of bile-pigment is to be detected by washing portions of the calculus and treating them with a diluted solution of potash. A deep-yellow color indicates the presence of bile-pigment, which may be determined by Gmelin's test, the addition of impure nitric acid.

The cholesterin calculi have a white color and crystalline structure, while the pigment-lime calculi are homogeneous. The largest calculi are found in the gall-bladder, and are composed almost entirely of cholesterin.

Biliary calculi may be found in the gall-bladder by the thousand (Otto). They vary in size from that of a pin-head to that of a hen's egg. The more numerous they are, the smaller are they. Their shape is round, oval, or oblong, and they are often facetted as a consequence of pressure. They are sometimes found elongated, their opposed surfaces so rounded and hollowed as to form jointed surfaces. In color they vary from the white of cholesterin to the brown and almost black of pigment-lime. Cholesterin calculi have a firm consistency, but are readily cut like wax, while pigment-lime calculi are brittle and are easily crumbled when dry.

They may be found anywhere in the biliary tract from the duodenal papilla to the minutest bile-ducts. The canal in which they lie may be dilated, perhaps sacculated. The mucous membrane may be smooth and shining, or, in case of inflammation, thickened, ulcerated, or perforated. In the latter case a suppurative inflammation around the gall-bladder or common or cystic duct may be present, with adhesions to neighboring organs or parts. Perforation may take place either into the peritoneal cavity or into the organs or parts adjacent. In the former case death is likely to rapidly follow, while in the latter fistulous communications are established with the duodenum, stomach, transverse colon, right renal pelvis, ureter, abdominal wall, or through the diaphragm with the bronchi, and the calculi may be discharged through the channels thus established.

Suppurative inflammation of the biliary tract may be connected with the gall-stones and extend into the gall-bladder, producing an empyema, or into the hepatic ducts, producing a cholangitis. The cholangitis when acute may result in abscesses of the liver by direct extension or by the production of a pylephlebitis, which may directly result in abscesses, or indirectly by embolism. If the cholangitis be chronic, it may produce a local cicatrix, or may, when diffuse, result in a fibrous hepatitis. The possibility of biliary calculi serving as a local cause for cancer of the gall-bladder or common duct has been frequently urged from their not uncommon association—according to Musser in 69 per cent. of the cases of primary cancer of the gall-bladder.

Symptomatology.—Gall-stones may be present and produce no symptoms; they may also, when sufficiently small, pass through the common bile-duct without giving rise to any symptoms. When the latter are present, they are due to expulsion, impaction, or incarceration, and the presence of the calculus is first suggested by an attack of biliary colic or by the occurrence of symptoms indicative of an inflammatory process in the region of the gall-bladder. It is claimed that lesser degrees of disturbance—discomfort in the region of the liver, slight enlargement, and slight jaundice—may be due to gall-stones, but such symptoms are no necessary evidence of their presence.

The gall-stone usually announces its existence when it begins to move from the gall-bladder into the cystic duct, or through the common duct into the duodenum. Kraus states that this usually takes place two or three hours after eating, especially at night, perhaps from improper food and drink or insufficient exercise. The symptoms which characterize this journey are pain, vomiting, fever, enlargement of the liver, and jaundice. The pain is usually sudden, severe, rapidly increasing in intensity. It is referred to the epigastrium, from which it radiates in all directions, frequently into the right shoulder and arm.

The pain is cutting or stabbing, tearing or grinding, and often causes the patient to writhe in his agony. Pressure gives no relief, and is often resisted by the tension of the abdominal rectus. It may last for hours, days, or weeks, with occasional remission and intermission, or it may suddenly cease. The pain may be simulated by that of hepatic neuralgia. The latter is less exactly localized to the region of the gall-bladder, occurs in nervous persons, and is without fever and usually without jaundice. It frequently alternates with other forms of neuralgia. Soon after the onset of the pain retching and vomiting begin, and are persistent despite the ejection of the contents of the stomach. The vomitus contains bile in case the common bile-duct be not obstructed by the calculus, and may sometimes contain gall-stones.

Chills or rigors, followed by elevated temperature in the course of a few hours, soon follow the attack of pain. The temperature may be as high as 104° or 105° F., and may rapidly fall if the colic be of short duration, or may last for several weeks in case inflammation of the biliary tract follow the attempt at expelling the calculus. If the fever be prolonged, it is likely to assume a remittent or intermittent type, resembling that of malaria. The pulse is often slow, from 40 to 50, but may be feeble, frequent, and irregular.

The abdomen may be somewhat retracted from contraction of the rectus. It is sensitive in the region of the liver, the lower border of which may extend to the level of the navel. The enlargement of the liver may subside in a few days. If it persist for a longer time, it is suggestive of an acute cholangitis.

The gall-bladder may be sensitive and tender, or it may be contracted and hidden below the surface of the liver. If it remain persistently sensitive and tender after the subsidence of the colic, it is probably inflamed.

The spleen may be enlarged, though it is not frequently so.

Jaundice is present in about one-half the cases, and begins within a few hours after the onset of the pain. It usually disappears from the skin within a week after the disappearance of the pain, unless the stone becomes impacted or incarcerated in the common duct, when it may persist for months. The stools are constipated, and may be colored or colorless, or may be colored and colorless alternately. They may be colored in the presence of jaundice. These variations in the color of the stools depend upon the varying degrees of obstruction of the common duct by the calculus from time to time.

During the attack of pain the patient's expression is anxious, his movements are restless, hiccough is not infrequent. The skin becomes cold and clammy, perhaps cyanotic.

Course.—The length of the attack is always uncertain, and may last for hours or days. Its termination is represented by the cessation of pain and the disappearance of fever. Swelling of the liver and jaundice may last a few days longer.

From the frequency with which many gall-stones are present it is not unusual for several short attacks of biliary colic to take place within the course of a few months, many months then elapsing before another paroxysm occurs. Several attacks may again take place, to be followed by another intermission. The immediate attack may be the last, or it may be followed by others of like nature, or it may result in impaction with chronic jaundice, dilatation of the bile-ducts or gall-bladder, or inflammation of the biliary tract with its results.

If the gall-stone does not escape through the common duct, it may become impacted either in this duct or in the cystic duct. If impacted in the common duct, it gives rise to persistent and progressive jaundice. In time the ducts behind the calculus may become dilated, or a diverticulum containing the calculus may be formed. It is then possible for the flow of bile to be resumed for a longer or shorter time, and the stools to be colored. The jaundice may persist for months, and the stones eventually escape or become encapsulated within a diverticulum from the duct. More often in the course of a year the symptoms of cholæmia set in, with marked delirium, convulsions, coma, and death.

If the impaction be in the cystic duct, the gall-bladder becomes distended, and is to be recognized as a palpable tumor which may extend below the navel. The gall-bladder may become inflamed, and a painful distention with febrile and digestive disturbances, follow.

If the gall-stone be impacted in the cystic duct, this tube may become obliterated in front and behind the stone; if incarcerated in the gall-bladder, a chronic inflammation of this organ may take place, resulting in its transformation into a fibrous capsule enveloping the calculus. This capsule may eventually be infiltrated with lime salts and form a bone-like shell.

The gall-stone which be incarcerated in the biliary tract may serve as a cause of acute inflammation of the bile-ducts. Frequent chills, followed by febrile paroxysms, attacks of mild jaundice, nausea, loss of appetite, constipation, emaciation, are evidences of this condition which may result in multiple abscesses of the liver and is likely to end fatally. Similar symptoms of pain and tenderness in the region of the gall-bladder are evidences of an inflammation of this organ, which may result in perforation into the peritoneal cavity, rapidly fatal, or in circumscribed adhesive peritonitis, with the formation of biliary fistulæ, usually associated with prolonged invalidism and symptoms of septicæmia.

Diagnosis.—The sudden, severe, radiating epigastric pain, quickly followed by retching and vomiting, enlargement of the liver, and perhaps by jaundice, are sufficiently suggestive of the attempt of the gall-stone to pass through the cystic or common ducts. The pain and vomiting are independent of indigestible food, as in case of acute gastritis, nor do they immediately follow the ingestion of food, as in ulcer of the stomach. After the passage of the gall-stone the pain is immediately relieved, and there is no prolonged convalescence with the necessity of a carefully arranged diet.

The probable diagnosis is made absolute by the discovery of the gall-stone in the diluted fæces strained through a fine sieve. This search if negative in its results should be continued for a number of days after the colic has ceased.

The pain and vomiting may resemble those occurring in the gastric crises of tabes, but they are not preceded by the lightning pains, and are not associated with the failing knee-jerk of that disease. Neither in this affection nor in lead-colic is there any enlargement of the liver, while in the latter affection the occupation of the individual and the blue alveolar line are suggestive of the nature of the ailment. Renal colic may be equally sudden and severe, but the pain is seated in the lumbar region and extends along the course of the ureter toward the bladder or into the penis or testicle. The presence in the urine of blood, albumin, and gravel explains the nature of the attack.

In pancreatic hæmorrhage or in acute pancreatitis the patient is usually fat, the collapse is greater, and the epigastric tenderness is more marked and extends into the splenic region. The pain and vomiting are less persistent, and sensitive points (from fat-necrosis) are likely to make their appearance in the abdomen. The fever persists and the stomach remains irritable. In peritonitis from perforation the abdomen rapidly becomes tense and tympanitic, the vomiting is less uncontrollable, and the fever is continuous. In appendicitis the pain is easier checked, and the tenderness is found in the region of the appendix. In catarrhal jaundice the initial symptoms are less severe, and the jaundice and digestive disturbances persist for a longer period.

In rare instances the symptoms of repeated biliary colic may be present without the discovery of a gall-stone and without palpable evidence after laparotomy of the presence of gall-stones. Such attacks are usually designated as hepatic neuralgia—a term which, though questionable, may be conveniently employed for this class of cases for want of a more exact knowledge of the conditions present.

If the gall-stones fail to pass, and obstruct the common duct, persistent jaundice becomes the prominent symptom. The sudden onset of the jaundice opposes the existence of a tumor or rupture of the duct, while the absence of cachexia and enlargement of the liver negative the theory of a cancer. If the gall-stone obstruct the cystic duct, enlargement of the gall-bladder and the absence of jaundice are the important symptoms.

The inflammatory disturbances connected with the presence of gall-stone in the liver pursue the course of a suppurative hepatitis, the cause of which is made known by the antecedent history of biliary colic. In the early stages of this process the suggestion of intermittent fever is often presented. Antiperiodic doses of quinine, however, are not effectual in preventing the repetition of the chills. If the inflammatory disturbances are in the gall-bladder, as an empyema or as a circumscribed peritonitis, the history of biliary colic again becomes important in the differential diagnosis.

The source of biliary fistulæ is to be recognized by the appearance of the gall-stone; and intestinal obstruction by a gall-stone follows the course of an intestinal colic, with a previous history of an inflammatory process in the vicinity of the gall-bladder.

Prognosis.—An individual may have many annual attacks of biliary colic or he may die in his first attack. Death during the attack is more likely to take place from collapse in an enfeebled patient—for instance, one with a fatty heart. Death from cerebral hæmorrhage has occurred during the attack. Rarely a fatal peritonitis may follow rupture or perforation of the biliary tract. The discovery of the gall-stone in the intestinal contents has a certain value in prognosis, as a facetted calculus is one of many. Between two and three hundred have been found in the fæces in less than a week. Every fæcal evacuation for at least a week should be diluted with water, stirred, decanted or poured through a fine sieve, that no concretion of the size of a pin-head may escape recognition. Those which pass through the common duct are usually not larger than a pea. Failure to find a concretion is important, as suggesting that the calculus is too large to pass, and may prove a cause of future disturbance. Failure to find the calculus is no necessary evidence of its retention, since, as Naunyn's experiments show, the calculus may be decomposed in the intestine. Frerichs reports a case where the patient recovered after seven months of persistent jaundice.

Treatment.—The rational treatment of gall-stones is based upon the theory that they are caused by an inflammatory process in the biliary tract and by obstruction to the flow of bile. The former is to be guarded against by the avoidance of highly seasoned or indigestible food and abstinence from alcoholic excess. Especially is such care to be exercised by the individual who has already experienced an attack of biliary colic. Measures which promote the flow of bile are care in partaking of only easily-digested food, abundant exercise, regular dejections. If the person has already suffered from biliary colic, the exercise should be such as increases the action of the diaphragm and abdominal muscles—*e. g.* climbing mountains and riding horseback.

Morning draughts of hot water, Vichy or Carlsbad, are to be recommended. The salicylate of sodium promotes the discharge of a thin, watery bile, and may be used freely diluted with water to the extent of 15 grains daily in case of suspected gall-stone. Tablespoonful doses of olive oil, twice daily, have been extensively used within the past few years with possible benefit. Continental authorities favor Durande's remedy:

℞. Olei terebinthinæ, ℈iv.
Ether, ʒv.
M. Sig.—Fifteen to thirty drops three times daily,

the action of which apparently promotes peristalsis, and thus favors the flow of bile.

There is but one indication when the attack of biliary colic has begun—to relieve pain. This is to be accomplished by the subcutaneous injection of morphine, or morphine and atropine, and by the inhalation of ether or chloroform. The injections are to be repeated at intervals of fifteen minutes until the severity of the pain is controlled. Hot poultices and fomentations are also advisable, and frequent drinks of hot water may be used. It should be remembered that the pain may cease suddenly with the escape of the stone, and after the intensity of the pain is relieved the morphine should be given at longer intervals, to avoid its poisonous action.

If the attacks of biliary colic are becoming frequent, or the gall-stone has become impacted or is producing severe inflammatory disturbances, surgical relief should be advised as the means of securing exemption from suffering by the removal of the gall-stones, and of saving life by preventing possible abscess of the liver, general peritonitis, or progressive cholæmia.

TUMORS OF THE BILIARY TRACT.

The benign tumors of the biliary tract, the fibroma and myxoma of the gall-bladder, are so rare as to demand no especial consideration. Cancer may be present both in the gall-ducts and in the gall-bladder, and is probably of more frequent occurrence than is generally recognized.

Etiology.—Cancer may be present as a primary or secondary growth, in the latter instance being extended from cancer of the liver, stomach, pancreas, or peritoneum.

Primary cancer is usually found in persons beyond middle life, in both sexes—in men less often than in women (Musser)—and is frequently associated with gall-stones. It may be found in the bile-ducts, usually at the duodenal end of the common duct, less often near the junction of the cystic and hepatic ducts. It is much more often found in the gall-bladder than in the gall-ducts, Musser having recently collected 100 cases of the former and but 18 of the latter.

Cancer of the Gall-ducts.

Morbid Anatomy.—Cancer of the gall-ducts usually appears as a circumscribed elongated infiltration of the submucous tissue and of the deeper layer of the mucous membrane, near the duodenal papilla. It apparently arises within the mucous membrane, since the surface of the latter is often intact.

Symptomatology.—It produces stenosis and dilatation of the common duct, a chronic jaundice of extreme intensity, associated with complete absence of bile from the intestines. Naunyn states that in about one-half of the cases of chronic jaundice cancer of the biliary tract is present, It is likely to prove fatal in the course of three or four months from cholæmia before ulceration or extension to other parts occurs. An associated cholangitis may produce the irregular fever with jaundice mentioned in the consideration of the latter affection.

Diagnosis.—An absolute diagnosis is practically impossible. Its presence may be inferred from a rapidly progressive and persistent, intense jaundice, without repeated attacks of biliary colic.

Treatment.—The treatment is symptomatic, and is to be directed more particularly to the relief of the jaundice.

Cancer of the Gall-bladder.

Cancer of the gall-bladder starts as a circumscribed elevated thickening of the wall near the fundus or near the cystic duct. As it progresses the entire wall of the gall-bladder or a large portion of it may become thickened from the cancerous infiltration. The new formation may be medullary or fibrous, its surface nodular or papillary. The gall-bladder may be increased in size to that of a child's head, partly from the growth of the cancer, partly from associated fluid. It is more commonly of the size of the fist, with an irregular surface; it may be shrivelled or contracted. The inner surface is often friable and shreddy from degeneration and necrosis of the growth, and the cavity of the gall-bladder contains an opaque gray or yellow, often bile-stained, fluid, in which bits of the growth may be found, and in which gall-stones are likely to be present. The cancer may be continued directly into the liver, or may extend to the portal fissure and invade Glisson's capsule. It may be found in the portal lymph-glands, and may penetrate the portal vein and give rise to multiple nodules in the liver. The secondary nodules in the liver may be numerous and much more voluminous than the original disease. Adhesions may be found between the gall-bladder and the colon or duodenum, and fistulous communications be established.

Symptomatology.—Cancer of the gall-bladder may be wholly unsuspected until a laparotomy or post-mortem examination has revealed the lesion. The early symptoms are essentially those of a cholelithiasis. There may be no jaundice until late in the disease, when it is usually present. A rapidly progressive cachexia, associated with symptoms of gall-stones, should suggest the diagnosis. The enlarged gall-bladder may compress the duodenum, producing vomiting, perhaps hæmorrhage, thus suggesting a tumor of the pylorus.

The physical examination may reveal a distended and sensitive gall-bladder, which may be resistant and nodulated or smooth and fluctuant, with rapid increase in size. Secondary nodules may project from the surface of the liver. Aspiration of the enlarged gall-bladder is likely to procure a bloody fluid, an abundantly albuminous bile, or an opaque yellow fluid containing a granular detritus.

Diagnosis.—The diagnosis is difficult in those instances where a palpable tumor does not exist, and is based upon a rapidly progressing cachexia, associated with symptoms of biliary colic and a slight or absent jaundice. When complicated with ascites, the absence of an enlarged spleen is conspicuous.

The **course** is usually rapid, and the prognosis eventually fatal, death occurring within a few months after the disease is recognized.

Treatment.—The treatment is symptomatic. The surgical treatment of this affection cannot as yet be said to hold out much hope of cure, although from the early limitation of the disease to the gall-bladder extirpation of the organ at this period might prolong life and add to its comfort, even if it produced no more permanent benefit.

JAUNDICE (ICTERUS).

Jaundice, or icterus, is the term applied to a yellow discoloration of the skin and many of the tissues and fluids of the body. It is a symptom of frequent occurrence in many diseases and under a variety of conditions. It is usually associated with a group of symptoms attributed to the action of the acids of the bile.

Etiology.—Two varieties of jaundice are usually recognized. The one, hepatogenous, with obstructed bile-ducts and abundant bile-acids in the urine, is due to the presence of bile-pigment, and results from the absorption of bile through the lymphatics of the liver in consequence of the obstructed outflow of the secretion.

The other, hæmatogenous, with open bile-ducts and absence of bile-acids in the urine, is attributed to the presence of blood-pigment, which is set free by the blood without the mediation of the liver. The theory of a jaundice from suppression of secretion has been effectually disposed of by Stein, who found that jaundice failed to occur when the entire blood-supply of the liver (of pigeons) had been completely cut off.

The agencies productive of hæmatogenous jaundice are found in certain diseases, as typhoid and yellow fevers, scarlatina, malaria, septicæmia, anæmia, and icterus neonatorum. Certain poisons, as ether, chloroform, phosphorus, arsenic, chloral, chlorate of potash, that of mushrooms, decayed fish, and snake-venom are also capable of producing it.

Especial support has been given to the view of the occurrence of a hæmatogenous jaundice by the experiments of Ponfick and others, who found that jaundice occurred when a certain proportion of the hæmoglobin of the blood was set free, either by its solution or by the disintegration of red blood-corpuscles.

Stadelmann, on the contrary, claims that there is but one variety of jaundice, the hepatogenous. His experiments explain the occurrence of jaundice under the circumstances above mentioned, on the ground that the agents used by him in freeing hæmoglobin in the blood produced a plugging of the small bile-ducts by a viscid bile or a catarrhal secretion, or caused their compression by producing a swelling of the liver-cells or leucocytic infiltration of the interstitial tissue. He further found that bile-acids were present in the urine, thus giving evidence that bile was absorbed.

According to this view, which may be considered to prevail at present, all jaundice is obstructive, and is due to the absorption of bile from the bile-ducts. The obstruction may affect the ducts within or without the liver. The causes of obstruction are as follows:

- Of the extra-hepatic ducts (the common and hepatic ducts).
  - Internal causes.
    - Inflammation.
      - Catarrhal.
      - Suppurative.
    - Stenosis.
      - Congenital.
      - Acquired.
    - Tumors.
      - Fibroma.
      - Lipoma.
      - Cancer.
    - Foreign bodies.
      - Gall-stones.
      - Fruit-seeds.
      - Parasites.
        - Distomum.
        - Lumbricus.
        - Echinococcus.
  - External causes.
    - Cicatricial tissue.
      - Duodenal ulcer.
      - Peripylephlebitis.
    - Fæcal retention.
    - Aneurism.
      - Aorta.
      - Hepatic artery.
      - Mesenteric artery.
    - Tumors.
      - Gall-bladder.
      - Duodenum.
      - Colon.
      - Pancreas.
      - Portal lymph-glands.
      - Omentum, mesentery.
      - Kidney.
      - Uterus (including pregnancy).
      - Ovary.
    - Parasite.—Echinococcus.

- Of the intra-hepatic ducts.
  - Internal causes.
    - Inflammation.
      - Catarrhal.
      - Suppurative.
    - Calculus.
    - Concretion.
    - Impacted bile.
  - External causes.
    - Fatty infiltration.
    - Passive congestion.
    - Inflammation.
      - Parenchymatous.
      - Interstitial.
      - Suppurative.
      - Tubercular.
    - Tumors.
      - Cancer.
      - Sarcoma.
    - Parasite.—Echinococcus.

Morbid Anatomy.—The color of the skin varies between a pale lemon-yellow and a deep yellowish brown. Shades of green and blue may be recognized, and a bronzed appearance may suggest the existence of Addison's disease. These differences in color depend upon the quantity of pigment in the tissues, the thickness of the superjacent epithelium, and the quantity and quality of blood in the superficial vessels. The skin is the darker in color the more complete the obstruction and the longer the duration of the jaundice. The pigment lies in greatest abundance in the deeper layers of the epidermis. All the tissues of the body are more or less deeply stained, with the exception of the teeth, cartilage, and nerve-tissue. Sweat and milk become bile-stained, while tears, saliva, and mucus are unstained. The fibrinous sputa of acute pneumonia, on the contrary, are pigmented. Of the internal organs, the liver and kidneys are most deeply stained, becoming of a dark olive-green color. The liver-cells are diffusely stained or contain granules or flakes of pigment. The capillary bile-ducts are frequently filled with elongated and branched plugs of inspissated bile. The larger bile-ducts may be dilated and filled with dark viscid bile, or their walls may be thickened and ulcerated and their contents be a thin, gruel-like fluid.

The search for the cause of obstruction should begin at the duodenal end of the common duct, where it may be found as a thickened mucous membrane or a mucous plug. If the large bile-ducts are free from obstruction, a microscopic examination of the smallest bile-ducts becomes necessary.

In the kidneys the pigment is found as a diffuse stain and in flakes: the latter may be present in the epithelial lining or in the canal of the tubules.

Symptomatology.—The symptoms due to an obstructed outflow of bile depend entirely upon the abnormal distribution of the pigment and of the acids of the bile. Within three or four days after the obstruction has taken place the discoloration of the skin and conjunctivæ is evident. It is usually earliest recognized in the latter, where it is made more conspicuous by contrast with the still unstained portions of white sclerotic.

The urine may be of a dark-brown color, resembling porter. It foams readily, and the froth is of a yellow color. A similar color may result from the use of rhubarb and santonin, but there is then no yellow foam and the color becomes red on the addition of caustic potash. The urine is of normal quantity, acid, clear, without albumin, and with but little sediment. In the latter are urates, bile-stained renal epithelium, and hyaline casts.

Gmelin's test is the one usually employed for the detection of the bilirubin. It consists in the production of a play of colors, in which green is characteristic, by the oxidation of bilirubin with impure nitric acid. The acid may be allowed to flow down the side of a wineglass containing an equal quantity of urine, or a few drops of acid and urine may be allowed to unite in thin layers in a white flat dish. The play of colors is to be seen at the line of confluence of the acid and urine. Rosenbach suggests that the urine be filtered and a drop of nitric acid be spread on the filter-paper. Brücke adds a few drops of nitric acid to the urine until it becomes green, and allows concentrated sulphuric acid to flow to the bottom of the glass. The play of colors then takes place at the border between the acid and the urine.

The reaction may be ill-defined or may take place but slowly, especially in the urine of fever and in stale urine.

Rosin has lately advocated the use of a modification of Maréchal's test with iodine. One part of tincture of iodine is added to ten parts of alcohol. A moderately thick layer of this mixture is allowed to flow gently over a portion of the suspected urine in a test-tube or wineglass. If bile be present in the urine, a ring of a grass-green color is formed at the line of apposition.

The search for bile-acids usually involves too much detail to be of general clinical value. They may be rapidly decomposed in the blood, and be found only in traces in the urine, and the urine in health has been found to contain them. Strassburger's modification of Pettenkoffer's test is usually employed for their recognition. After isolation, cane-sugar is added to the extract, which is then filtered. A drop or two of strong sulphuric acid is spread on the dried filter; a violet or purple color appears.

Normally the bilirubin is rapidly transformed in the bowels into urobilin, and the fæces, when deprived of bile-pigment, are pale, clay-colored, dense, and offensive. They frequently contain, especially when the diet is fatty, clusters of acicular crystals, regarded by Œsterlen as a magnesia-soap. They may be pale from a milk diet, and yet contain bile, and may be dark-colored and free from bile when iron or bismuth are present or when there has been intestinal hæmorrhage.

The absence of bile from the intestines interferes with the absorption of fat and favors putrefaction. Flatulence and constipation are present, and tympany and colic may occur. There may be no disturbance of gastric digestion, or the tongue may be coated, the breath fetid, and the taste bitter. The disturbances of gastric digestion are more likely to be the result of the causes of the jaundice than attributable to the latter itself. In such cases there are loss of appetite and nausea. The patient complains of epigastric fulness after eating and of emptiness before meals. The area of hepatic dulness may be increased at first, and the region of the liver may be tender and painful, and the gall-bladder may be distended.

The pulse is usually lowered, varying from 40 to 60 per minute, although it may be found as low as 20 per minute. According to Legg, the slowing of the pulse is due to the action of the bile-acids on the cardiac ganglia.

The respiration is usually unaffected. The temperature is normal, except in the presence of inflammatory complications. Both temperature and pulse are then elevated, but range lower than from the same causes in the absence of jaundice.

There may be headache and vertigo. The patient is likely to be irritable or despondent, his mind may be dull or stupid, or he may be wide awake, even to sleeplessness.

The vision may be curiously disturbed, some patients seeing better by obscured light—nyctalopia; others suffering from unusually obscured vision as darkness approaches—hemeralopia. To some, objects appear yellow—xanthopsia, a symptom so rare as not to be attributable to the presence of bilirubin in the eyes.

Itching of the skin is frequent, and may form a most distressing symptom. It is attributed by some to the presence of bile-pigment in the skin, by others to the irritating effect of bile-acids. The itching usually is not apparent until the skin has become discolored. It is worse at night and in bed, and may be universal or be most intense in the palms and soles, between the fingers and toes. Scratching results in papules, pustules, ulcers, and crusts. Urticaria, herpes, boils, and carbuncles may be present, and hæmorrhages may take place in the skin as well as elsewhere. Xanthelasma has been observed in the more chronic forms of jaundice.

Course and Duration.—Acute and chronic varieties of jaundice are recognized, the former lasting for a few weeks, the latter perhaps extending over a period of years, Fagge reporting a case lasting seven years. In the chronic cases there may be periods of exacerbation and remission, but the longer the duration the more likely are permanent and serious organic changes to be present—*e. g.* hypertrophic cirrhosis and atrophied liver. Death may result with profound disturbances of the nervous system, as restlessness, delirium, and coma. The symptoms of grave import are gastro-intestinal hæmorrhages, elevated temperature, and disturbance of vision. An apparently mild case may suddenly develop the gravest symptoms of acute yellow atrophy.

Diagnosis.—In recognizing the presence of jaundice it is important to appreciate the influence of race in the coloration of the skin. In Addison's disease the sclerotic is not pigmented, and the discoloration extends over a long period: the head, hands, and flexures of the body are more deeply stained than other portions. In jaundice both skin and urine are discolored, and the urine contains bile-pigment. In determining the cause of jaundice the seat of the obstruction should be considered. If at the duodenal end of the common duct, the fæces are colorless and the gall-bladder may be distended. If in the hepatic duct, the gall-bladder is not distended and the fæces are colorless. The paler the fæces and the more acute and intense the jaundice, the more likely is the obstruction to be outside of the liver. If the fæces are bile-stained and the skin jaundiced, the obstruction is more likely to affect certain of the bile-ducts within the liver.

Most acute, uncomplicated cases of jaundice are probably catarrhal. If associated with fever, the catarrh has probably extended into the smaller ducts. If associated with pain in the region of the gall-bladder or with attacks of biliary colic, gall-stones are the probable cause, especially if the attacks are recurrent.

Chronic jaundice, if mild, is most likely to result from fibrous hepatitis, malignant disease of the liver, or chronic passive congestion of this organ. The coexistence of ascites points to the first of these causes ; of hepatic enlargement and deformity, the second ; and of chronic mitral disease, the third.

Prognosis.—The prognosis depends upon the cause. If readily removed, the stools become colored, the pigment leaves the skin, and finally disappears from the urine. Catarrhal jaundice usually lasts from two to six weeks, yet acute yellow atrophy of the liver begins as a catarrhal jaundice. Chronic jaundice, especially if increasing in severity, should always excite suspicion of a more serious disease than catarrh of the bile-ducts or gall-stones.

Treatment.—The treatment of jaundice relates to the removal of the cause and to the relief of the symptoms. The usual cause is a swollen mucous membrane or a viscid secretion at the duodenal end of the common duct. Its removal takes place spontaneously with the subsidence of the inflammation, and demands only a suitable diet and regulation of the bowels. If the cause be an impacted or incarcerated gall-stone, its removal can only be effected by surgical measures or by the establishment of a fistula.

The diet should be free from fat and not readily putrescible. Skimmed milk, strained broths, egg, with but little farinaceous food, may be given at first. As improvement takes place, lean meat, chicken, fish and vegetables may be used, and cooked acid fruits may be eaten. Water, lemonade, Seltzer, Apollinaris, or soda-water may be used as a drink. The tendency to constipation is best relieved by small daily doses of a saline laxative, as Epsom and Carlsbad salts. Krull has recommended the rectal injection of one to two quarts of cold water.

Itching should be treated with calomel ointment, lotions of carbolic acid, lead-water, and glycerin or diluted hydrocyanic acid. Internal remedies may also be necessary, as bromide of potassium or sodium, or morphine.

Sleeplessness not due to itching may be relieved by sulphonal or chloral.

When the flow of bile into the intestines is resumed, remedies may be employed to increase its secretion and promote its flow. Such are aloes, colocynth, rhubarb, or podophyllin, the sulphate of potash, and sulphate or salicylate of sodium. The last drug is especially indicated when the obstruction is seated in the small bile-ducts, as it favors the discharge of a liquid bile.

Warm baths promote the desquamation of the skin and the removal of pigment from the surface. The removal of bilirubin through the kidneys is promoted by the use of saline drinks, especially Carlsbad, Vichy, and Lithia water. The daily use of the bitartrate or acetate of potash will accomplish the same purpose.

ACUTE INFECTIOUS JAUNDICE (WEIL'S DISEASE; BILIOUS TYPHOID).

In 1886, Weil reported a series of cases in which jaundice was associated with a splenic tumor and symptoms of nephritis as a peculiar infectious disease. Since then numerous publications on the same subject have appeared, especially in Germany and France, and a general agreement has been reached as to the grouping of the symptoms under this head, although it is by no means apparent that these symptoms have the same etiology; and, until the latter is more clearly established, clinical convenience is served by the use of the term Weil's disease or of acute infectious jaundice.

Etiology.—The disease affects males almost entirely, who are for the most part between the ages of fifteen and thirty years, and may occur in isolated instances or as small epidemics. It is of more frequent occurrence in summer, and is usually encountered in regions or localities where there is faulty drainage or where persons are exposed to the influence of putrescent material. These factors have been so constantly present as to be regarded as the cause of the symptoms, but it is likely that there may be several causes.

Morbid Anatomy.—In the few post-mortem examinations that have been made nothing characteristic has been discovered. A granular, in part fatty, degeneration of the cells of the liver and kidneys has been found, also foci of leucocytic infiltration, in these organs.

Symptomatology.—The disease usually begins unexpectedly with a chill, followed by fever. The temperature rapidly rises to 104° or 105° F., and remains elevated until between the fifth and eighth days, when it falls by steps, becoming normal between the tenth and twelfth days. The pulse ranges between 100 and 110, but falls with the onset of jaundice. Headache and vertigo are early symptoms, and are followed by restlessness, delirium, stupor, and prostration, the latter being sometimes extreme. There are severe pains in the nape of the neck, back, and especially in the calves. The tongue is coated; there are loss of appetite, nausea, and perhaps vomiting. Jaundice makes its appearance early, is mild, and lasts perhaps a fortnight. About the seventh day cutaneous eruptions are frequent—roseola, erythema, perhaps itching and herpes. Epistaxis, sore throat, or bronchitis may be present.

With the onset of the jaundice the liver is likely to be enlarged and tender and the spleen is enlarged. The urine contains albumin, hyaline and epithelial casts, red and white blood-corpuscles. There may be diarrhœa or constipation, and the stools are pale, sometimes colorless, during the jaundiced period.

In about one-fourth of the cases a recurrence of the fever takes place within a week following the return to the normal temperature and the alleviation of the symptoms. The recurrent fever is milder than the original attack, and may last five or six days.

The convalescence is slow, extending over a period of two to three months before the strength is fully restored. There are but few complications, among which parotitis and peripheral neuritis may be mentioned.

Diagnosis.—The nature of the symptoms is indicative of an infectious process, and the alimentary tract is thought to be the usual channel of admission. The diagnosis only becomes clear with the advance of the disease.

It has been regarded as an abortive typhoid fever, a bilious form of relapsing fever, and the anatomical changes have been claimed to be identical with the typhus icterodes of Egypt. Weiss, one of the most recent writers on the subject, considers that the symptoms and lesions thus grouped together resemble closest the bilious typhoid fever described by Griesinger.

Prognosis.—The prognosis is usually favorable. The rare fatal cases may occur early or late in the disease.

Treatment.—The treatment is symptomatic. following the lines laid down for the treatment of typhoid fever. The cerebral excitement and muscular pains are likely to demand the use of soporifics and anodynes.

DISEASES OF THE PANCREAS.

BY REGINALD H. FITZ.

General Symptomatology.—The especial symptoms which are usually considered as suggestive of disease of the pancreas, are fatty stools, lipuria, and glycosuria. These are all symptoms which have been found in connection with disease of the pancreas, as well as in other affections. The first attracted more attention in a previous generation than at present, and the last has become of renewed and extreme importance since the experiments of Mering and Minkowski.

The pancreatic juice is known to possess four distinct digestive properties: 1, the emulsifying of fat; 2, the saponification of fat (Müller); 3, the saccharifying of starches; 4, the peptonizing of albuminoids. All of these properties have been carefully studied with reference to their value in diagnosis and treatment. The first two are conspicuously related to the phenomenon of *fatty stools.*

The early medical writings contain occasional references to the presence of fat in the stools, and Elliotson in 1833 refers to them in his paper on this subject. The fat may be present in solid masses, compared in size with walnuts, grapes, beans, and peas, opaque white or translucent yellow, resembling tallow or butter, firm or soft, greasy to the touch, and melting by heat or burning like tallow. Liquid fat may appear in the stools, lying upon the fæces like melted butter, burning with a bright flame, and becoming solid when cool, or the alvine discharges may be at times wholly composed of oil.

On microscopical examination the fat is found (Müller) as acicular crystals (fat acids or combined with lime or magnesia as soap), or it is present as fat-drops or as irregular flakes. The fat acids form drops when heated, and are dissolved in ether, while the soap-crystals must first be treated with acid before heat will dissolve them.

It was early recognized that fat might be present in the stools if the diet were excessively fatty or if a large quantity of oil were swallowed. Olive oil in particular was claimed "to concrete into mucus when taken into the alimentary canal;" cod-liver oil and castor oil are known to behave in a like manner; and olive oil produces the pseudo-gall-stones which have been regarded as evidence of the benefits of sweet oil in promoting the escape of gall-stones. The association of the fatty stools with jaundice, and with the evidence of disease in the alimentary canal, the liver, and pancreas, was also observed.

In the past sixty years, chiefly owing to the communication of Richard

Bright, it has been the prevailing idea that fatty stools are a characteristic symptom of disease of the pancreas. He reported three cases of fatty stools; in each of the patients there was "disease, probably malignant, of that part of the pancreas which is near to the duodenum, and ulceration of the duodenum itself." He admitted that his reasoning with reference to the relation of the fatty stools to the diseased condition found afforded "but slender connection even to my [his] own mind." The communication was made as a hint to be improved by future observers. "Should future experience and observation serve in any way to connect the peculiar evacuation which is at present under consideration more decidedly with a diseased condition of the pancreas or an imperfect action of the duodenum, which would, in all probability, be associated with pancreatic derangement, it will possess much interest," etc., etc.

From this time on numerous illustrations of the association of fatty stools and pancreatic disease were published, and the suggestions from the clinical observations seemed to be supported by the statements of Claude Bernard, that the pancreatic secretion was the most important in the accomplishment of the absorption of fat.

It was in consequence of this association of clinical and experimental evidence that Friedreich (1875) admitted that the presence of fatty stools might be utilized with caution in the recognition of an affection of the pancreas, especially when there were other reasons in favor of such a disease, and when there was sufficient certainty that a simultaneous disease of the liver might be excluded. Friedreich admitted that they may occur in health from an excessive introduction of fat into the stomach, and that they may be present when there is no disease of the pancreas; furthermore, that they are often absent when there is disease of the pancreas.

The early evidence of their diagnostic value thus depended upon their presence in assumed uncomplicated pancreatic disease with suppression, obstruction, or qualitative alterations of the secretion, and upon the claim that the pancreatic secretion is essential to the absorption of fat.

Satisfactory evidence of the first of these propositions is lacking, and the experimental evidence of the relation of the pancreas to the absorption of fat is contradictory. On the one hand, it is claimed that it is essential to the absorption of fat; on the other, that fat may be absorbed despite the absence of pancreatic juice.

The former view is based upon the experiments of Bernard, and more recently upon those of Mering and Minkowski, who found that when the pancreas was extirpated free fat appeared in abundance in the stools. Abelmann also found that after extirpation of the pancreas all fat given appeared in the fæces. If only a portion of the pancreas was removed, less than 60 per cent. of the fat taken was absorbed.

The latter view is advocated more particularly by Müller. It is recognized that the bile and duodenal secretion may emulsify fat. He found that when the flow of bile into the intestine was completely cut off, the fæces contained from one-half to three-fourths the amount of fat taken into the stomach.

This result occurred whether the obstructive jaundice was due to gall-stones, cancer, or catarrh of the common duct. Friedreich had also recognized that fatty stools were especially frequent and copious where there was conjoint disease of the liver and of the pancreas.

Müller also claimed that the saponifying of fat was a peculiar property of the pancreas, was shared in only by the intestinal bacteria, especially by the colon-bacillus, and that the pancreatic juice possessed this quality in much greater degree than did the bacteria. The presence of saponified fat in the fæces would therefore be suggestive of absent pancreatic disease. This view, however, is contradicted by Abelmann's later observation, that where the pancreas was extirpated saponified fat and free fat-acids appeared in the fæces, and represented the greater part of the fat taken into the stomach of the animal.

It would therefore appear that free or saponified fat in the fæces is no necessary evidence of deficient pancreatic secretion, since they may be absent where there is obstruction to or failure of pancreatic secretion, and may be present where there is no pancreatic disease—not only when there is an excess of fat in the food, but also when there is a deficiency of bile, even in the absence of jaundice, as shown by Berggrün and Zuntz in the chronic peritonitis of children. Furthermore, the stools may be fatty when the absorbing apparatus is diseased, as in amyloid or tubercular disease of the intestine, chronic enteritis, and disease of the mesenteric glands.

Although Mering and Minkowski found that starch was for the most part discharged undigested from the animal deprived of the pancreas, the saccharifying of starch is a property of the saliva as well as of the pancreatic juice, and the failure to saccharify a certain amount of starch would have no diagnostic value, if for no other reason than the difficulty of its ready recognition at the bedside.

Much more important, whether due to the absence of pancreatic secretion or to the failure of the animal to consume the sugar in the organism after the loss of his pancreas, is the occurrence of *diabetes in pancreatic disease.*

The concurrence of pancreatic disease and diabetes was first noted by Cowley in 1788, and has been repeatedly observed since his time, especially since Lanceraux's observations in 1877. Friedreich justly claims that the diabetes may be secondary to the pancreatic disease, or that disease of the pancreas may be secondary to the diabetes. The lesions of the pancreas with which diabetes was found were atrophy, fatty degeneration, suppurative and fibrous inflammation, concretions, cysts, and tumors.

Experimental proof of the origin of diabetes from pancreatic disease has been recently furnished by Mering and Minkowski (1889), who showed that after the complete removal of the pancreas from dogs all the characteristic symptoms of severe diabetes occurred. The essential features were polyuria, polydipsia, polyphagia, and rapid loss of flesh and strength. These experiments have since been frequently repeated, with like results, by a variety of observers, and furnish satisfactory proof that complete destruction of the pancreas or its function in certain animals causes diabetes. Incomplete extirpation fails to produce diabetes.

In human pathology like differences are met. In Trafoyer's case the sequestrated pancreas was discharged from the bowels, and the patient was living and well seventeen years later. It may be that a portion of the pancreas remained. Otis reports the successful removal of the pancreas through an abdominal wound in the War of the Rebellion. Here, again, the possibility of a portion of the pancreas being left behind is obvious. Bull, on the contrary, operated upon a patient with a pancreatic cyst who subsequently died of diabetes. The occurrence of grave diabetes should therefore direct attention to disease of the pancreas as a possible cause, although diabetes often occurs without disease of the pancreas, and disease of the pancreas often occurs without diabetes.

The *peptonizing of albuminoids* is a property of the pancreatic secretion shared by the gastric juice. It was observed, however, by Mering and Minkowski that after extirpation of the pancreas an abnormally large quantity of undigested muscle-fibres appeared in the fæces when a diet largely of meat was provided. It has been suggested that the diagnosis of pancreatic disease with failing or obstructed secretion might be confirmed by the presence of abundant undigested muscle-fibres, provided there was no diarrhœa, which would tend to force them through the bowel with undue rapidity.

In fluid supposed to come from a pancreatic cyst, on the contrary, the peptonizing qualities may be so slight that large quantities are necessary for its determination. The absence of peptonizing qualities is therefore no satisfactory reason for rejecting the pancreatic origin of the fluid.

It is claimed by Pisenti that the pancreatic juice changes proteids into peptones, and these into leucin and tyrosin, from the decomposition of which skatol, phenol, and indol arise, the last being the source of indican. An obstruction to the flow of pancreatic secretion into the bowel should thus diminish the quantity of indican in the urine. Under normal conditions the quantity of indican in this secretion is but small. It is, however, increased in tumors of the stomach and intestines. If, then, according to Leo, an epigastric tumor should be associated with a normal or diminished quantity of indican in the urine, this tumor would be rather pancreatic than gastric or intestinal.

Still another test of failing pancreatic secretion, according to Nencki, is furnished by the action of salol. When this drug enters the bowel it is decomposed by the pancreatic juice into salicylic acid and phenol. The latter is eliminated by the kidneys as a sulpho-carbolate, and if abundant produces the familiar dark-brown, carbolic discoloration of the urine. A drachm of salol given in divided doses through the day is sufficient for this purpose. The failure of the color reaction is evidence of lacking pancreatic secretion.

Brief allusion may be made to a few additional symptoms which have been supposed to hold a more or less intimate relation to disease of the pancreas. The first of these is *lipuria*.

The possibility that fat in the urine might prove a symptom of disease of the pancreas was suggested by the case reported by Elliotson of the passage of liquid fat from the bowels and in the urine. No post-mortem examination was held in this case; hence no satisfactory evidence of pancreatic disease was obtained.

Clark (1851) observed lipuria and fatty stools in a case of cancer of the pancreas combined with nutmeg liver, and Bowditch (soon after) reported a case of cancer of the liver and pancreas in which lipuria occurred. The possibility of lipuria as a symptom of pancreatic disease is made greater in the light of recent experiments from the observation of its occurrence in diabetes. It is recognized that the blood of diabetic patients may contain an abnormal quantity of fat, and Kobert had a patient who periodically passed fat and sugar in his urine, both ingredients disappearing when the diet was restricted. The symptom is so rare, however, as to be of but little value.

Another symptom is *emaciation*, which may result from pancreatic disease, but is a symptom of so many diseases as to have no diagnostic value. When present in pancreatic disease, it may be the result of associated lesions of the stomach, intestines, liver, or other organs. Pancreatic disease may also occur in fat persons. After closure of the duct or extirpation of a considerable part of the pancreas there may be no emaciation. Even after removal of the human pancreas by operation (Otis) or by sloughing (Trafoyer) the patient may remain well.

Salivation is another symptom of assumed importance from its occurrence in a few cases, none, however, of uncomplicated pancreatic disease. It was explained on the ground of sympathy or of vicarious function. It is usually absent in pancreatic disease and present in gastric affections, and is no evidence of the former.

Watery dejections were supposed to occur in some forms of disease of the pancreas, and were sometimes called *pancreatic diarrhœa.* Their occurrence was assumed to result from irritation of the pancreas. Since mercury irritated the salivary glands and caused loose movements, the latter were thought to represent a like irritation in the pancreas, the abdominal salivary gland, which might occur from other irritants.

Bronzed skin and *cœliac neuralgia* were sometimes noted in pancreatic disease, but oftener in its absence. The former is more particularly identified with Addison's disease, and the latter occurs in so many affections as to have no diagnostic significance.

PANCREATIC HÆMORRHAGE.

The occurrence of hæmorrhage within and near the pancreas has attained great importance within recent times, since it has been discovered as the sole important lesion in certain cases of sudden death.

Attention was first conspicuously called to this lesion and its results by Zenker in 1874, and Prince and Draper subsequently added to our knowledge of this subject.

Etiology.—The immediate cause of pancreatic hæmorrhage is unknown. It has been suggested that the corrosive action of the pancreatic secretion might be of influence, or that injury, as a blow upon the abdomen, might be of importance. It is also possible that the hæmorrhage may be of nervous origin, since nodules of pulmonary hæmorrhage, in the absence of embolic causes, have been found associated with the pancreatic hæmorrhage. The persons affected have usually been adult males of more than forty years of age. Several have been intemperate, others were abstemious. It may be said, in general, that there is nothing in sex, habits, conditions, or exposure which makes pancreatic hæmorrhage a likely occurrence.

Morbid Anatomy.—The hæmorrhagic infiltration is to be found in the gland and in the subperitoneal tissue in its vicinity. In the latter case the hæmorrhages may be continued into the fat-tissue of the omentum and mesentery, behind the colon, or around the kidney. The hæmorrhage is usually diffuse, but it may be in numerous patches both beneath the peritoneal investment and in the interlobular tissue. The entire pancreas may be infiltrated, or either half or the central portion alone may be conspicuously involved. The pancreas may be of normal size or enlarged; the density may not be altered, or the gland may be flaccid, soft, and friable. Its structure may be normal, or the epithelium may be granular, or the pancreas may be fatty infiltrated or fatty degenerated.

Symptomatology.—Pain is frequently an early symptom, either severe or intense, and is referred to the epigastrium, to the lower part of the chest, or to the abdomen. Pain, however, may be absent or slight, and there may be merely a sensation of constriction of the chest. Nausea or vomiting occasionally occurs, and the bowels may be constipated or there may be a frequent desire to stool.

The most constant symptoms are those of collapse, and are more or less intense and more or less prolonged. The collapse is usually explained, in accordance with Zenker's suggestion, by a reflex paralysis of the heart, as occurred in Goltz's experiment of the blow upon the abdomen of the frog.

Result.—Death is not infrequent, and may take place within an half hour, or it may result after several hours. It is probable that recovery from lesser degrees of hæmorrhage may occur, since evidences of antecedent hæmorrhages have been found in a case of recent fatal bleeding. It is also probable that inflammation of the pancreas may be induced by the hæmorrhage, and the symptoms and lesions of this condition be produced and be continued over a longer or shorter period. It is also probable that cysts of the pancreas or a circumscribed peritonitis of the lesser omental cavity may be a result, which may end in recovery, perhaps, in consequence of surgical interference.

Treatment.—The only treatment to be recommended for pancreatic hæmorrhage is by stimulants during the period of collapse, opiates for the relief of pain, and a subsequent treatment, either medical or surgical, as a pancreatitis, pancreatic cyst, or hæmorrhagic peritonitis is developed.

ACUTE PANCREATITIS.

In the present elementary state of our knowledge concerning acute pancreatitis it seems desirable to consider each anatomical variety apart, although the hæmorrhagic and gangrenous varieties probably represent the same conditions etiologically, while the suppurative variety may have a different etiology, as it presents a different clinical picture.

HÆMORRHAGIC PANCREATITIS.

This term is applied to the concurrence of pancreatic hæmorrhage and evidences of an acute inflammation of the gland.

Etiology.—It has already been stated that cases of pancreatic hæmorrhage may end in hæmorrhagic pancreatitis. The etiology of the two affections may thus be common. The importance of trauma in the production of hæmorrhagic pancreatitis has been recently illustrated by the occurrence of a case in the wards of Dr. J. Collins Warren at the Massachusetts General Hospital. The patient was brought to the hospital intoxicated and in a state of collapse. He had been run over by a wagon, and died at the end of thirty-six hours. Several of the lower ribs had been broken, the liver was torn, and hæmorrhage had taken place in the body of the pancreas near the tail. There were multiple foci of fat-necrosis, and the microscopic examination of the pancreas showed a cellular infiltration of the interstitial tissue. Most of the cases thus far reported have been adult males, from twenty-five to sixty years of age, more than half of whom have been abundantly or superabundantly provided with fat-tissue. About the same proportion were persons who had suffered from previous digestive disturbances, rather gastric or gastro-duodenal than enteric, and, as in Day's case, from attacks of biliary colic. A small fraction was addicted to the abuse of alcohol. The conspicuous factors in etiology are, therefore, previous attacks of gastro-duodenitis, alcohol, and injury. The gastro-duodenitis probably acts by direct extension of the inflammation along the pancreatic duct. Alcohol acts as a predisposing rather than as an exciting cause; while trauma acts directly.

Morbid Anatomy.—The pancreas is enlarged, either throughout or at one extremity, usually the head. It may be doubled in size, or may seem still larger when abundant fat-tissue is present. The gland may be dense or of diminished consistency and friable.

The presence of hæmorrhage is to be suspected by the color of the surface, which is of various shades of red. On section the color may be dark red, reddish-brown, or reddish-black. The shades of red may be uniformly distributed or may lie in specks or patches. The color of the section may be modified by an excessive quantity of fat-tissue, giving translucent yellow bands or spots mottled with shades of red. In addition, there may be opaque white specks, spots, or streaks—the fat-necrosis of Balser. The pancreatic duct may contain a bloody fluid, and its branches may be plugged with clotted blood. The splenic vein may contain a thrombus. The hæmorrhagic infiltration may be continued, as in pancreatic hæmorrhage, into the peripancreatic tissue and into the mesentery, meso-colon, omentum, and into the subperitoneal fat-tissue, between the descending colon and left kidney to the brim of the pelvis. The peritoneum usually shows no alterations, although evidences of recent peritonitis may at times be found, and subperitoneal patches of fat-necrosis are common.

On microscopical examination evidences of extensive hæmorrhagic infiltration are found, chiefly limited to the interlobular tissue. In the same region are found cellular and fibrino-cellular exudations. Round cells and red blood-corpuscles are also to be found in the acini and in the ducts. Many of the lobules present the appearance of a coagulation-necrosis and thrombi are to be seen in the smaller veins. Bacteria are present in large quantities in the inflamed gland. Osler and Hughes mention the presence of leucocytes in the semilunar ganglia and indistinctness and cloudiness of the ganglion cells.

Symptomatology.—The disease usually begins suddenly and unexpectedly, though sometimes preceded by some irregularity in diet, with abdominal pain. This pain may be violent, intense, or severe, constant or paroxysmal. It is usually seated in the upper abdomen, and sometimes follows the course of the pancreas. Eventually the localized pain may be extended to various parts of the abdomen, limited to the region of what eventually appear as foci of fat-necrosis, or diffused throughout the abdomen.

The pain is usually followed by vomiting, rarely by nausea alone. The vomiting is constant and repeated, or occasional, and may be copious. It may be bilious or black, and has contained dark-red masses as large as peas. Constipation is frequent and diarrhœa rare. Hiccough may be present, and delirium may occur.

The temperature may be subnormal on the second day, or slightly elevated, perhaps considerably raised. Slight degrees of collapse may be an early feature, while symptoms of collapse almost invariably precede death.

The upper abdomen not infrequently becomes swollen and tympanitic, and later there may be a general abdominal swelling and tympany. In a case of recent occurrence not only was there epigastric tenderness, but the intercostal spaces in the splenic region were sensitive to deep pressure.

In the rapidly fatal cases death takes place within the first three days, or the fatal issue may be delayed till the end of the first week. If the patient survive longer than this period, the case runs the course of a gangrenous pancreatitis unless recovery occurs.

Diagnosis.—The sudden and unexpected occurrence in a healthy individual of violent epigastric pain, and the subsequent appearance of vomiting, collapse, fever, and a circumscribed tympanitic abdominal swelling, are the characteristics of diagnostic importance. The differential diagnosis lies practically between an irritant poison, perforation of the digestive or biliary tracts, and acute intestinal obstruction. The history of the case and the examination of the vomit will exclude the theory of an irritant poison.

The absence of pain after eating, hæmorrhages from the digestive canal, and anæmia will exclude a gastric or intestinal ulcer. Acute perforation of the colon is rare, and the resulting peritonitis progresses more rapidly, and is likely to be present from the outset. Perforation from gall-stones is usually preceded by attacks of biliary colic and jaundice, while the seat of pain and tenderness is rather in the region of the gall-bladder than in that of the pancreas.

Acute intestinal obstruction is most likely to give rise to a doubt. It is to be eliminated by determining through injection the patency and capacity of the large intestine, by the infrequency of an obstructed small intestine in the epigastric region, by the localized tenderness in the region of the pancreas, and by the usual early absence of conspicuous general tympany or limited distention of intestinal coils. The suspicion of acute intestinal obstruction has repeatedly led to an exploratory laparotomy.

Prognosis.—Our knowledge of the prognosis of hæmorrhagic pancreatitis is largely based on the fatal cases. The occurrence of similar, less severe, symptoms at an early period in the patient's history, and the presence of crystalline blood-pigment and fibrous thickening, suggest that recovery from a mild attack may take place. This possibility is made almost a certainty by the case mentioned by Osler. The patient was supposed to have intestinal obstruction of three or four days' duration, and laparotomy was performed. No obstruction was found, but the peritoneal cavity contained a small quantity of bloody serum. There was a dense, thick, indurated mass in the region of the pancreas and at the root of the mesentery, and foci of fat-necrosis were found in the mesentery and omentum. The patient recovered.

Treatment.—The first indication is to relieve the patient's suffering by morphine subcutaneously and by the local application of heat. The symptoms of collapse are to be relieved by stimulants by the mouth or rectum. The subsequent treatment of the case is that of peritonitis with little or no exudation.

Gangrenous Pancreatitis.

Hæmorrhagic pancreatitis not terminating fatally or in improvement in the course of a week usually results in gangrenous pancreatitis.

Etiology.—Since gangrenous pancreatitis is to be regarded as one of the results of a hæmorrhagic pancreatitis, the etiology of the two affections is largely the same. As in the latter, so in the former—the most constant antecedents are more or less repeated attacks of digestive disturbance. These are attended with pain, usually referred to the region of the stomach or resembling biliary colic. They are frequently accompanied by vomiting, and not rarely by jaundice. In rare cases ulcer of the stomach and gall-stones may precede the affection of the pancreas.

The patients are adults, without especial distinction of age or sex.

Morbid Anatomy.—During the early stages of this disease the appearance of the pancreas resemble that of hæmorrhagic pancreatitis. The gangrene may be apparent as early as the fourth day, when the tip may be shreddy, or the entire gland be transformed into a dark, slate-colored, glistening mass. The adjacent parts may be infiltrated with a discolored purulent fluid, or the coils of intestine near the pancreas may be united together and to the under surface of the diaphragm by recent adhesions. The diaphragm may present a dirty, grayish-white, shreddy appearance, and be infiltrated with dirty-gray pus. As the gangrene progresses the pancreas may lie in a spongy meshwork infiltrated with green fluid. A section of the gland may show areas of hæmorrhagic infiltration, spots of softening, and opaque-white areas of fat-necrosis. Towards the end of the second week the pancreas may form a soft black, shreddy slough lying in the lesser omental cavity, attached by a few threads to the wall. The omental cavity may contain an abundant bloody, ichorous fluid, or may have emptied its contents through a perforation of the stomach or duodenum. If the patient survive, the lesser omental cavity may form a part of a large pus-cavity extending behind the cæcum and descending colon to the pelvis. The contents may be a greasy yellow detritus with masses of necrotic fat-tissue as large as a hen's egg, sequestrated from cavities in the wall, stained with dark-brown or iron-rust pigment. The sloughing pancreas may be discharged through the bowels as early as in the fourth week.

Disseminated fat-necroses are frequent. The spleen may be large or small, is often soft, and its vein plugged with a thrombus. The thrombus may extend to the portal vein, and may be puriform. A general suppurative peritonitis is rare, although adhesions between coils of intestine near the root of the mesentery are not infrequent.

The inflammatory process may extend to the pleura and pericardium; acute leptomeningitis may occur, and death may be the result of pulmonary embolism.

Symptomatology.—The initial symptoms are, as in hæmorrhagic pancreatitis, sudden, unexpected abdominal pain, often intense or severe, constant or paroxysmal. The pain has been referred to the stomach, left hypochondrium, navel, mid-abdomen, left loin, and back. The vomit consists of partly-digested food, or is viscid or slimy, green or black, sometimes bloody. Chills are occasional, sometimes at the outset, and fever is frequent in the course of a few days. The temperature is usually in the vicinity of 100° F., although it may rise as high as 104° F. Again, there may be no suspicious elevation of temperature throughout the disease.

Jaundice is occasionally observed after a few days. The urine may contain albumin and casts.

Abdominal swelling is often present, and usually occurs late in the disease. It may be slight or enormous, general or limited to the epigastrium or to the left half of the abdomen. The abdominal swelling is usually tympanitic, although dulness may be present in the flanks. The spleen is rarely palpable. The parotid gland may become swollen.

As the disease runs its course the characteristics of a subacute peritonitis are gradually developed, often limited to the upper abdomen, and seated in the lesser omental cavity, rarely extended to the general peritoneal cavity. The patient loses flesh and strength. Vomiting, diarrhœa, and abdominal pain are conspicuous, and death takes place, usually preceded by collapse.

Diagnosis.—The diagnosis of a gangrenous pancreatitis is based upon the recognition of a deep-seated peritonitis spreading downward from the epigastric region, perhaps simulating by its physical characteristics a cyst of the pancreas. The development of the swelling is preceded by the symptoms suggestive of a hæmorrhagic pancreatitis.

Prognosis.—Although the result has been almost invariably fatal, death taking place in the course of three to eight weeks, recovery has occurred. The products of the circumscribed peritonitis and the sloughing pancreas have been discharged through the bowel, and in Trafoyer's case the patient was alive seventeen years later.

Treatment.—The treatment of gangrenous pancreatitis is that of a circumscribed peritonitis, and is distinctly surgical. It should be undertaken before perforation of the stomach or intestine has taken place from the cavity in which the pancreas lies. The escape of pancreatic sloughs through the wound gives the most satisfactory evidence of the nature of the process. In the meantime, until the circumscribed peritonitis has made itself apparent, the medical treatment is that of hæmorrhagic pancreatitis—symptomatic and sustaining.

SUPPURATIVE PANCREATITIS.

Acute pancreatitis not ending in resolution and not associated with hæmorrhage or gangrene, is likely to terminate in suppuration. There is nothing in the etiology of the original attack in virtue of which the suppurative termination is to be anticipated. Previous attacks of indigestion and errors in diet are of greater importance than traumatism, and the latter factor is not recognized in the etiology.

Morbid Anatomy.—The appearances of the pancreas vary in accordance with the severity and persistence of the attack. The pancreas may be decidedly enlarged, reddened, and studded with innumerable small abscesses. Many of these may break through their peritoneal covering, and be associated with the appearances of a fibrino-purulent peritonitis. In the more chronic cases a solitary abscess, perhaps as large as a hen's egg, may be present, with extension of the suppuration to the peripancreatic tissue, or several smaller abscesses may be seen connected together and discharging into the adherent stomach or duodenum. In the most chronic cases the abscess in the pancreas may empty into the lesser omental cavity, which becomes distended and forms a large circumscribed abscess lying behind the stomach and intestines. The cavity may communicate with the duodenum. A peritonitis limited to the vicinity of the pancreas is frequent.

In suppurative pancreatitis fat-necrosis is rare; thromboses of the splenic and portal veins are not infrequent. Abscesses of the liver may be associated. The spleen is usually not much enlarged.

Symptomatology.—Sudden severe, usually intense, gastric, epigastric, or abdominal pain, vomiting, and sometimes great prostration, may announce the attack. At about the third day a slight elevation of temperature occurs, and the epigastrium is likely to become swollen, sensitive and tympanitic. Hiccough and chills may take place, and a moderate general tympanitic distention of the abdomen is likely to occur. Such cases, few in number, run a course similar to that described in Hæmorrhagic and Gangrenous Pancreatitis, and prove fatal in the course of a week or more. With similar violent symptoms at the outset the disease may be prolonged over some six or seven weeks. During the greater part of the time there may be frequent irregular chills and atypical, often high, temperature. Slight jaundice may be present and pains in the hypochondria, with enlargement of the liver and spleen. There may be exacerbations and remissions in the course of this more prolonged disturbance, which may be interrupted during the third or fourth week by violent paroxysms of lancinating pain starting laterally in the epigastrium, perhaps followed by temporary collapse and continued diarrhœa, with subsidence of symptoms. This condition has been found to result from an emptying of the abscess into the stomach and duodenum. In these cases death may occur in the course of one or two months from progressive emaciation and debility, the symptoms being those of septicæmia.

In other instances of suppurative pancreatitis the early symptoms may be less severe. The patient gradually becomes weak and thin. There may be little or no fever or there may be periods when chills and fever are present, and there may be obscure signs of peritonitis. There is but little pain, although there is a frequent sense of distention of the stomach, especially after food is taken. There is no appetite ; diarrhœa may be present, or the stools may be consistent, colorless, and offensive, especially when there is slight jaundice. If the abscess breaks into the stomach or duodenum, the stools may be bloody. In the course of months anasarca or ascites is likely to occur, and death may take place in the course of six months or a year. In these chronic cases a bronzed skin and saccharine urine have been observed.

The **diagnosis** of this variety is dependent upon the recognition of a deep-seated subacute epigastric peritonitis.

Prognosis.—All known cases have proven fatal within a few weeks or within from six to twelve months. The early recognition and the appropriate surgical treatment offer hopes of a smaller mortality in the future.

MULTIPLE DISSEMINATED FAT-NECROSIS.

The frequent mention of this condition in connection with acute pancreatitis makes a more detailed consideration desirable.

Although minute foci of necrosis are occasionally found in fat-tissue and in bone-marrow in wasting or in chronic diseases, and similar appearances, according to Balser, in swine, they are found in man in such number and distribution, and with such serious concomitants, as to give them a grave pathological importance.

Etiology.—Extensive disseminated fat-necrosis, with inflammatory and hæmorrhagic phenomena, are found almost invariably in connection with pancreatic disease alone. Rolleston has recently recorded a case where the fat-necrosis was present and no lesion of the pancreas found. In a personal letter he states that Dr. Hawkins observed this condition in a man who died after symptoms of intestinal obstruction lasting four days. The pancreas presented no lesions visible to the naked eye. The fundus of the gall-bladder was ulcerated, and bile was present in the peritoneal cavity. In the great majority of cases, however, there is found simultaneous disease of the pancreas.

Balser, who first wrote in detail concerning this condition, regarded it as evidence of a disease which, when extensive, was associated with old and recent hæmorrhage. He stated that it might become so excessive in very fat people that a large part of the abdominal fat dies, a fatal result occurring on account of either the quantity destroyed or the associated hæmorrhage. More extensive observations, however, show that it may occur in lean as well as fat people, and that, even when extensive, it does not necessarily cause death. Neither is it always associated with any considerable hæmorrhage, nor is it invariably a cause of pancreatic hæmorrhage, since it may occur without this lesion. Although most frequently found in connection with hæmorrhagic or gangrenous pancreatitis, it may occur in the suppurative variety.

From the fact that disseminated fat-necrosis is usually found in connection with acute pancreatitis, and, at the outset, is limited to the immediate vicinity of the pancreas, it would seem as if the pancreatic affection might be a cause. In a case of recent occurrence it was most extreme in the immediate vicinity of the injured and inflamed pancreas, and became less extensive the greater the distance of the fat-tissue from the pancreas.

The chief feature in its gross appearances is the opacity of the affected fat-tissue, which is found to be due to the presence of fat-crystals, either of stearin, as shown by Dr. Harrington, or of fatty acids combined with lime, as shown by Langerhans. The suggestion is plain that contact with the pancreatic secretion may produce the crystallization. Cohnheim states that the pancreatic secretion from an artificial pancreatic fistula may flow into the abdominal cavity without injury to the animal experimented upon. Kühne, on the contrary, found that when a solution of trypsin, the ferment found in the pancreatic juice, was injected subcutaneously, the most extreme destruction followed. It is well known that pancreatic juice has the property of splitting fat into free acids and favors the formation of soap-compounds of fat acids and lime or magnesia. It seems not impossible that this result may occur in man under pathological conditions, and serve as the immediate cause of the foci of fat-necrosis. This view receives a substantial support from the recent successful experiment of Langerhans, who injected the minced pancreas of the rabbit into the subcutaneous tissue of another rabbit, with the production of fat-necrosis.

The foci of fat-necrosis also contain numbers of bacteria, Ernst having found four varieties and Welch one, the colon-bacillus. It has been shown that intestinal bacteria, especially the colon-bacillus, also possess the property of splitting neutral fats into free acids and compounds of them with lime and magnesia. It is hence possible that their presence in foci of fat-necrosis may be the cause of the crystallization of the fatty acids. Mention may also be made of the recent discovery by Balser of a fungus resembling the actinomyces in the necrotic foci of swine.

Morbid Anatomy.—The foci of fat-necrosis in acute pancreatitis are found in the subperitoneal fat-tissue of the abdominal wall, mesentery, and omentum, and in the fat within and around the pancreas. They have been found in the subperitoneal fat of the diaphragm, and Chiari has found them in the subpericardial, subpleural, and subcutaneous fat. They have also been found in bone-marrow.

They are usually present in great numbers, discrete or in masses. They are superficial or deep-seated, and vary in size from that of a pin-head to a hen's egg. They are more or less rounded, of an opaque-white or yellowish-white color, and are of a soft, tallowy consistency. The smallest nodules are often surrounded by an injected vascular wreath, while many are bounded by a distinct narrow reddish-brown zone, indicative of hæmorrhage. The larger nodules have been found incrusted with lime and surrounded by a dense fibrous capsule from which threads occasionally ran into the surrounding fat-tissue. The tallow-like material on microscopical examination is seen to be composed largely of acicular crystals in addition to fat-drops, and a granular detritus. The crystals are either stearin, according to Balser and Harrington, or combined with lime, according to Langerhans.

Within the patch of necrosis the outlines of the fat-cells may be distinct or they may be obliterated. At the periphery is often found a narrow orange-colored streak, in which are frequent granules and round clumps of small orange-colored crystals. Beyond them is a zone of round-celled infiltration. Within and around the foci of fat-necrosis colonies of bacteria may be found. In the surrounding fat-tissue are hæmorrhagic spots and clumps of leucocytes.

It is thus apparent that the earliest stage is one of crystallization of the fat, followed by an inflammatory line of demarcation and hæmorrhage. It is thus possible for the necrotic fat to be sequestrated, and, if this necrosis takes place about the pancreas, for this gland to be sequestrated and to hang to a few shreds or to be discharged through the intestine, as in the cases of hæmorrhagic and gangrenous pancreatitis.

The evidence is directly suggestive that the fat-necrosis is usually secondary to the pancreatitis, and is often an important element in the production of the hæmorrhage and in the extension of the gangrene and suppuration which are associated.

The disseminated fat-necrosis has an important practical side, since its discovery at a laparotomy leads to the recognition of the pancreatitis, and the course it pursues suggests the origin and course of the frequently associated peritonitis.

CHRONIC PANCREATITIS.

Chronic fibrous pancreatitis is a disease which is rarely diagnosticated, in virtue of its frequent association with disease of the digestive organs, and from the latency of its course. Although occasional striking illustrations of the occurrence of this affection are to be found in medical literature, it is not always clear that the cases so reported are not confounded with other diseases, especially with tumors of the pancreas.

Etiology.—In considering the causation of fibrous pancreatitis mention may be made of the presence of its anatomical changes in connection with suppurative processes. This event, the termination of suppurative pancreatitis in induration, has already been alluded to in the consideration of the former subject.

Genuine cases of fibrous pancreatitis are to be found in connection with a variety of conditions. One of the best recognized causes is syphilis. This disease is well known to produce a congenital pancreatitis, and infants afflicted with this lesion may live for months after birth. Whether or not extensive fibrous pancreatitis may be the result of acquired syphilis is doubtful.

The abuse of alcohol has been regarded as a cause of chronic pancreatitis. The evidence on this point is rather based on the analogous process in the liver than on satisfactory evidence of lesions of the pancreas due to this cause.

Extension of a chronic gastro-duodenal catarrh into the pancreatic duct is a not unlikely cause, especially in those cases where long-continued digestive disturbances have preceded death, and a fibrous pancreatitis is the sole significant lesion.

Chronic pancreatitis may also result from obstruction of the pancreatic duct; from extension of a chronic peritonitis from the neighboring peritoneum, perhaps in consequence of ulcers of the stomach or duodenum, tumors of the stomach, or suprarenal capsule, aneurism of the aorta or cœliac axis, and disease of the spine.

Lastly, there are the cases of indurated pancreas in connection with chronic obstruction to the circulation through the heart, lungs, or liver.

Morbid Anatomy.—The essential feature in the alteration of the pancreas is the increase of fibrous tissue. This may be distributed throughout the pancreas, giving rise to a symmetrical enlargement of the gland, or it may be limited to one part, especially to the head. In the latter case the enlargement may be such as to suggest a tumor of the pancreas. It is not unlikely that among the reported cases of scirrhus of the pancreas there are those due to fibrous inflammation, and not to cancer. The increase in the size of the gland may be as great as one-third in all the dimensions, the weight being doubled. The pancreas may also be diminished in size, on account of the shrinkage of the new-formed fibrous tissue. This change is especially likely to occur in chronic pancreatitis secondary to obstruction of the ducts, and in those cases where the increased fibrous tissue is extended from without inward.

The color may not differ from the normal, or may be more yellow or more white in individual instances. The consistency is increased, sometimes to such an extent that the density of the organ suggests that of cartilage. The surface is smooth or granular, and thickening and induration may be found at the root of the mesentery and in the vicinity of the suprarenal capsule and cœlic plexus.

On section the pancreas may present a more homogeneous appearance than normal, or granules of various size may be seen ; the projecting acini may be distributed throughout. These acini may be opaque-gray or opaque-yellow, the latter in case of associated degeneration. The duct may show no alteration of structure or be indistinct, or it may be dilated, tortuous, and ribbed, with corresponding atrophy of the glandular structure.

Klebs states that small white streaks or spots may be seen in the midst of the dense fibrous tissue, and that a sort of emulsion is to be squeezed from the spots, in which a granular, amorphous mass of molecules of lime may be present. He has also found clusters of radiating crystals of fatty acids, evidently from the atrophy of fat-tissue. The cheesy nodules, as large as peas, found by Drozda in the fibrous pancreas from a syphilitic patient, may have been gummata.

Symptomatology.—There are no characteristic symptoms of fibrous pancreatitis. As a rule, digestive disturbances, deep epigastric pain and tenderness, progressive emaciation, and debility are present.

The digestive disturbances are those usually attributed to gastric catarrh—viz., loss of appetite, nausea, belching, pyrosis, a sense of epigastric fulness and weight. These symptoms may precede for a long time the emaciation and debility which usually eventually occur. More rarely they may be wholly wanting. Vomiting is rare, and sometimes bloody, even in the absence of ulcer of the stomach. The bowels are irregular, diarrhœa being rather frequent. The stools may be colorless, are sometimes fatty even in the absence of jaundice. The latter symptom is occasionally present : it may be transitory or persistent, for the common bile-duct is sometimes compressed by the contracted head of the pancreas.

Epigastric pain, deep-seated, dull, burning, or boring, is frequently complained of. The pain may be paroxysmal, and, when severe, associated with great anxiety, restlessness, and attacks of faintness. The epigastrium, especially on the left, may be tender on deep pressure, and a sense of resistance, even a tumor, has been felt, either to the right of the median line or extending to the left and elongated. Slowly progressing and moderate ascites has been observed, and also a gradual enlargement of the spleen.

A symptom which has attracted attention in a number of cases is glycosuria, and the disease has run the course of severe diabetes.

The duration of chronic pancreatitis is uncertain. In some instances the above-mentioned symptoms precede death for a few months only, while in others the course may extend over a year or two. When glycosuria is conspicuous, it may be present four years before death. The experiments on rabbits by Arnozan and Vaillard show that a fibrous pancreatitis from ligature of Wirsung's duct may be well advanced within fourteen days after the ligature has been applied.

Prognosis.—The prognosis of chronic pancreatitis is necessarily grave. But little evidence can be obtained of a favorable result, since the diagnosis is always doubtful. Recovery, however, is not impossible or unlikely, since it is well recognized that patients may live for a long time after being deprived of a large part of the pancreas by operation, sequestration, and evacuation, by cystic degeneration or atrophy. Friedreich mentions the case of a patient in whom symptoms which might be attributed to chronic pancreatitis were recovered from.

Treatment.—The treatment would be that for diabetes in the glycosuric cases. The digestive disturbances would demand a diet largely farinaceous, and minced pancreas or pancreatin might be used. The latter are indicated in all cases of diminished pancreatic secretion, since Abelmann's experiments showed that the digestion of fat was promoted by their use when the pancreas was extirpated.

PANCREATIC CALCULI.

The discovery of calculi in the pancreas is usually regarded as an anatomical curiosity. At the same time, they are not infrequently associated with such distressing symptoms and serious lesions that they must be considered as possessing a high degree of importance in pancreatic pathology.

Etiology.—The only recognized factors of etiological importance in the production of pancreatic calculi are chronic inflammation of the duct and its obstruction. The existence of the former is inferred from the resemblance of the contents of the dilated duct to those frequently found in retained inflammatory products in mucous canals. The frequent association of obstruction of the duct and calculi admits of a two-fold explanation. The calculus is always found in a dilated duct, but a dilated duct does not always contain a calculus. Although the obstruction to the escape of the pancreatic secretion may be the result of an impacted or incarcerated calculus, it may be dependent on causes outside the duct, as a fibrous pancreatitis, a pancreatic or other tumor, or a pathological process producing compression or traction. In the secretion thus retained in the cavity salts may be precipitated.

Morbid Anatomy.—The calculi are composed chiefly of carbonate of lime, usually with more or less phosphate of lime, and may be found in any part of the pancreas, more frequently lying in the main duct alone or in it and its branches. There may be but one, or upward of a hundred may be present. The lime-salts may be present as a mortar-like material, or concretions may exist, varying in size from grains of sand up to masses as large as walnuts.

The shape of the smaller calculi is often round; the larger calculi are elongated, perhaps oval. They are not infrequently branched, and the surface, though perhaps smooth, is not rarely rough, even spinous. They have been compared to a piece of coral in virtue of the shape and character of the surface.

The color is usually either white or grayish-white. They are generally associated with various lesions of the pancreas, which, in part at least, may be considered as the result of their presence, in part as a co-effect of the common cause. The alterations usually found are dilatations of the duct, pancreatic abscess, chronic fibrous pancreatitis, atrophy, and rarely fistulæ of the pancreas extending into the stomach, duodenum, or peritoneal cavity.

Dilatation of the duct presents itself in a variety of ways, according to the seat and degree. If the smaller branches are dilated, they may project above the surface of the pancreas as rounded granules filled with a greasy pap, in which fat-drops, cholesterin crystals, and molecular lime may be present. The surrounding gland-tissue is atrophied; the fat-tissue may be increased or diminished. Klebs has applied the term "pancreatic acne" to this condition. The dilatation may affect chiefly the main duct, which becomes widened and tortuous, perhaps as large as the finger. The primary branches may be dilated, and the wall may present a series of valve-like projections. The fluid contents of the dilated ducts may resemble an emulsion of chalk, and the calculi may appear to be attached to the wall from their continuance into the smaller ducts. To this condition Virchow has applied the term "ranula pancreatica." Still greater degrees of dilatation produce the cyst of the pancreas.

The recorded cases of abscess of the pancreas in connection with calculi are open to doubt. They appear to represent localized dilatation of the duct, with agglomerated calculi and puriform contents, but do not give evidence of a wall of granulation-tissue. At the same time, a progressive destruction of tissue may occur with the presence of the calculus, as is shown by the perforation of the pancreatic wall and the discharge of calculi into the duodenum or stomach, with the establishment of a gastro-pancreatic or pancreatico-duodenal fistula. A fatal peritonitis from perforation into the peritoneal cavity has also thus arisen. The association of chronic fibrous pancreatitis and calculi is common. The pancreas is usually atrophied from the shrinkage of the new-formed fibrous tissue, although atrophy of the pancreas in connection with the calculus may occur without a marked increase of fibrous tissue. In rare instances pancreatic calculi are associated with cancer of the pancreas.

Symptomatology.—Calculi may be present in the pancreas without giving rise to any symptoms attributable to their presence, or they may be associated with the severest symptoms. When the latter occur, it is in general questionable whether they are due to the concretions or whether they and the concretions are not due to a common cause.

Most frequent among the early symptoms connected with calculi are those of disturbed gastric and gastro-duodenal digestion. In the majority of instances they run their course without the involvement of the pancreas. In most cases there are no especial symptoms to indicate when the pancreas is affected. On the other hand, the occurrence of deep-seated pain, in the region of the pancreas, and rarer paroxysmal attacks of intense pains resembling those of biliary colic, have been connected with the calculus. The paroxysmal attacks are due to the presence of the calculus near the outlet of the pancreatic duct, and may be associated with jaundice from resulting pressure on the outside of the common duct. Furthermore, biliary calculi are not rarely associated with pancreatic calculi. Although paroxysmal pain is likely to be a symptom in some cases of pancreatic calculi, it has no value in differentiating such cases from those of ordinary hepatic colic due to gall-stones in the common duct.

Symptoms of greater value in the history of pancreatic calculi are the progressive, sometimes extreme, emaciation and debility not infrequently observed. When these are associated with fatty stools and glycosuria, and in the absence of jaundice, pancreatic disease is directly suggested. Its calculous nature is to be suspected when constant or paroxysmal attacks of pain are associated. The latter eventually cease, perhaps to be followed by the development of a cystic tumor in the epigastrium. When a calculous pancreatitis is associated with diabetes, excessive appetite and thirst become conspicuous symptoms. The duration is essentially chronic, extending over a period of years. The fatal termination may be sudden, as in case of perforation.

Diagnosis.—A diagnosis of pancreatic calculus might be made, provided paroxysmal attacks of pain, simulating biliary colic, are associated with progressive extreme emaciation and debility, glycosuria, and absence of jaundice.

Prognosis.—The outlook in cases of pancreatic calculi is always uncertain, and is essentially that of the associated lesion. It therefore corresponds to that of chronic pancreatitis, pancreatic cysts, and pancreatic diabetes.

CYSTS OF THE PANCREAS.

This term, originally applied to a lesion rarely recognized as an anatomical curiosity, and considered to be due to the dilatation of Wirsung's duct, has been extended, especially in the past decade, to include a number of lesions characterized by the accumulation of fluid in the region occupied by the lesser omental cavity. A study of the cases thus recorded has led to the conclusion that the term is often used on insufficient evidence, and that a variety of cysts of the pancreas other than those from dilatation of the duct of Wirsung may occur.

Etiology.—Typical cases of pancreatic cyst may originate from dilatation of Wirsung's duct. Such dilatation may result from an obstruction of its outlet by tumor or stricture or valvular folds, and by the impaction of calculi. Cysts of small or large size may thus arise, and the latter may eventually produce serious disturbances. The dilatation may affect smaller branches of the main duct, and single or multiple cysts varying in size, the latter eventually becoming confluent, may thus arise. Rare instances of pancreatic cyst are to be found where the structure of the wall is suggestive of a cystoma, analogous to the ovarian cystoma.

The history of many recorded cysts of the pancreas is suggestive that the extension of an inflammatory process from the duodenum into the duct of Wirsung may lead to the obstruction of the latter, and thus result in a retention-cyst.

Traumatism plays an important part in the origin of circumscribed accumulations of fluid in the region of the pancreas, although these accumulations may prove to be rather the result of an encysted peritonitis of the omental bursa than a cyst within the pancreas. Such peritonitis may represent the extension from the pancreas of an acute inflammatory process to its peritoneal covering, which is the posterior wall of the lesser omental cavity.

Cysts of the pancreas occur with nearly the same frequency in men as in women. If all the accumulations of fluid near the pancreas were included among the cysts of this organ, the frequency would be found greater in men.

The affection is one of adult life, being equally common in the decades from thirty to forty and from forty to fifty years. It is about half as frequent between twenty and thirty and between fifty and sixty years.

The possibility that a pancreatic cyst may be of congenital origin is suggested by Richardson's case of the extirpation of a cyst containing seven pounds of sligtly opaque fluid from a child of fourteen months. The tumor was present at birth, and was attached to the tail of the pancreas, which was spread over the cyst-wall; in the latter pancreatic tissue was present.

Morbid Anatomy.—Cysts of the pancreas may be divided into monocysts and polycysts, and may arise within the head, body, or tail of the gland. They are globular, may reach the size of a pregnant uterus at full term, and have a smooth or trabeculated wall, not infrequently with circumscribed thickened portions forming slightly elevated patches. Secondary cysts may be found to communicate with the main cyst, and papillary excrescence may project from its interior. The lining-membrane is smooth and thin and covered with stunted cylindrical epithelium. In typical cases from dilatation the duct may be traced from the duodenum into the cavity of the cyst, and from the tail into the same cavity. At times the duodenal end of the duct is obliterated in the immediate vicinity of the cyst-wall.

The contents of the largest cysts may be as great as fourteen quarts, and are either a more or less viscid or watery alkaline fluid of a grayish color, slightly opaque, with a specific gravity of 1010–1024. On microscopic examination of the fluid, leucocytes, fatty degenerated epithelial cells, free fat, cholesterin, and acicular crystals may be found. This fluid usually presents some or all of the characteristics of pancreatic juice—viz., the power to emulsify fat, to transform starch into glucose, and to digest albumin and fibrin. The last quality is often lacking; the first two are usually present. These physiological reactions, however, are not sufficient to characterize the fluid as pancreatic. Von Jaksch and Boas claim that other fluids may possess diastasic and emulsifying qualities, while the peptonizing property may be absent or so slight that large quantities of fluid may be necessary to determine its presence. The older the cyst the less likely is the fluid to present all the above reactions.

Conspicuous attention has been called to the presence of blood in the contents as evidence of the pancreatic origin of the cyst. In most of the cases of so-called pancreatic cysts of traumatic origin fresh and decolorized blood-corpuscles, and flakes of blood-pigment or masses of blood-clot have been found. The tendency of pancreatic lesions to become hæmorrhagic is well recognized, but the presence of blood in cysts in the vicinity of the pancreas is more likely to occur when the pancreatic duct is not dilated than when such is the case.

Typical pancreatic cysts may be found without hæmorrhagic contents, and blood has been frequently found in circumscribed collections of fluid in the vicinity of the pancreas whose walls have no continuity of structure with this organ. Blood in the fluid is therefore of little or no diagnostic importance.

Multiple cysts of the pancreas may occur as retention-cysts—*e. g.* the cases reported by Klebs, Kuhnast, Martin, and Gussenbauer—or as cystomata, as reported by Salzer and Hartmann. In Kuhnast's case the body of the pancreas was made up of cysts, one as large as a medium-sized apple; all the cysts were filled with blood in all stages of transformation, and the dilated pancreatic duct was to be traced here and there between the cysts. In Martin's case the tumor was of sixteen years' duration, and held fifteen litres of fluid. A portion of the wall contained large and small cysts, from the size of a hazelnut to that of a goose-egg. Their contents were either a thick brown fluid or blood, in part fresh, or detritus. The duct was not found. In Gussenbauer's case the left half of the body and the tail of the pancreas were replaced by a collection of cysts, the largest of the size of a hen's egg, and containing a clear, slightly viscid fluid. The duct was open from the duodenum to the cyst.

On the border-line between the multiple retention-cysts and the cystomata are the cases reported by Riedel and Salzer. In Riedel's case the cyst was of nine years' duration, and contained ten litres of brown fluid. Four days after extirpation of the main cyst the patient died, and a small part of the otherwise intact pancreas was found tied very carefully with catgut. The head of the gland was easily recognized, but the body was lost in the wall of the cyst, the tail not being recognized. There were adenoid projections from the wall of the cyst. These globular cavities, lined with cylindrical epithelium, communicated with the cavity of the cyst, and were lined with papillary elevations, as in the ovarian cystomata. In Salzer's case there were connected with the cyst-wall two flat globular masses as large as a hen's egg, with a lobulated surface. On section they were found to be composed of cystic spaces filled with thick, slimy, grayish-white contents.

The possibility that the cystomata of the pancreas may be of a malignant character is suggested by the case reported by Hartmann. The tumor was of three months' duration and contained two hundred grammes of a brown fluid resembling chocolate. The patient died five weeks after the operation, and the entire body and tail of the pancreas were transformed into a mass of cysts between which the duct was to be traced. The cysts contained a viscid gray fluid. They were called cystic epithelioma by Gilbert, who found a new formation of glandular culs-de-sac, with cystic dilatation, as in cystic epithelioma of the breast. Cancerous nodules were found in the liver.

As the pancreatic cyst, whatever may be its origin, enlarges, it tends to form a large main cyst, which pushes toward the stomach and thus chiefly lies in the lesser omental cavity. It may cause atrophy of the greater part of the pancreas, or it may project from the gland as a pedunculated tumor. Its anterior surface may become fused with the posterior wall of the stomach, thus rendering extirpation difficult if not impossible. It may rupture into the omental cavity or into the general peritoneal cavity or into the interior of the stomach. The rupture of a small cyst into the lesser omental cavity may lead to a peritonitis of this sac, in which case, as the tumor projects toward the stomach, the latter organ is usually found overlying the upper portion of the same. More rarely the tumor may project above the stomach, the latter being displaced downward. The transverse colon may cross the cyst in front, or be displaced downward and be behind the symphysis in the case of a large tumor. The tumor lies more to the left side of the median line when it arises from the tail of the pancreas, or it may project so far to the right as to be mistaken for a distended gall-bladder. As it increases in size it may be limited to the upper abdomen, usually the greater portion of its bulk being found in the left hypochondriac and umbilical regions, or it may extend across the median line and fill the entire front of the abdomen as far as the brim of the pelvis. The inflammatory contents are likely to be mixed with pancreatic fluid, and may partake of its properties.

Symptomatology.—There may be no symptoms preceding the appearance of the tumor, or none due to its presence until it has attained a large size. The tumor may be accidentally discovered in a person in good health, or after childbirth or convalescence from typhoid. Usually the patient suffers from attacks of pain in the upper abdomen, which may radiate from the vicinity of the ensiform cartilage downward or to one side or the other, often to the left. More rarely the pain may shoot into the left shoulder or into the left half of the face. These attacks of pain may be sudden and severe, with symptoms of collapse. They may last for hours, days, or weeks, and may be continued at more or less frequent intervals for a period of years before the appearance of the tumor. Again, the tumor may be noticed within a period of three weeks after an attack of sudden pain and vomiting. The attacks of pain may occur unexpectedly or they may follow an error in diet; they may be accompanied by vomiting, belching, diarrhœa, or constipation; and there may be a sensation of epigastric fulness or tenderness, or of both. Jaundice may follow the attacks of pain, and intestinal hæmorrhage has been stated to have occurred at intervals preceding the appearance of the tumor. The appetite may be unaffected, diminished, or voracious. There may be little or no disturbance of nutrition or strength, or the patient may become emaciated and debilitated.

With the appearance of the tumor the symptoms usually become more serious. The pain and digestive disturbances are more constant, and symptoms suggestive of intestinal obstruction may be present. The larger the tumor the more pronounced is the emaciation and weakness, the patient sometimes being unable to make any active exertion. The pressure of the tumor then gives rise to dyspnœa and œdema. The urine rarely contains sugar or albumin and the stools are rarely fatty. An excess of undigested muscular fibres has sometimes been found in the fæces.

The tumor is the most important clinical characteristic of the pancreatic cyst. It lies in or near the epigastrium, and causes protrusion, at first, of the upper part of the abdomen. It usually appears in the left hypochondrium, between the costal cartilages and the median line ; more rarely it is felt in the vicinity of the navel. It is globular, resistant, not elastic, smooth, usually changing its position somewhat with the movements of the diaphragm, and possessing a slight degree of lateral motion. It often transmits the pulsation of the aorta, but has no expansile pulsation. When deep-seated it gives no sense of fluctuation, but as it nears the surface a wave is readily transmitted. It is dull on percussion, and on auscultation a systolic souffle has been heard, transmitted from the underlying and compressed aorta.

According to its size, its boundaries will vary. It is usually recognized before its lower border extends beyond the umbilical line, but it has been known to extend from the ensiform cartilage to the pubic symphysis and into each flank.

The smaller the tumor the more likely is it to suggest a solid growth, as it is deep-seated and the fluid is often under great tension, spurting several feet when a trocar is forced through its wall. Its relation to the stomach should be appreciated, since the cyst has been punctured through the overlying stomach in more than one instance.

Course and Duration.—Cysts of the pancreas are usually of slow growth, in one case the condition having been present for twenty years. They may remain at a standstill for a period of years—eight in one instance—and may then increase much in size within a few months. They are stated to have attained their maximum size within a fortnight, and in a single case to have suddenly become as large as a child's head. So sudden a method of enlargement is suggestive of hæmorrhage into the cyst.

The reported cases of the traumatic variety of pancreatic cysts almost invariably lack the kind of evidence which enables the diagnosis to be unquestioned. In no such instance has a cyst been extirpated. In the case reported by Lloyd, an enormous encysted hæmatoma was found behind the stomach and removable with it. It appeared as if the blood had become encysted between the posterior peritoneal coat of the stomach and the peritoneal covering of the pancreas. Lloyd calls attention to the probability of the traumatic cases of so-called pancreatic cyst being effusions into the lesser omental cavity and not cysts of the pancreas. Their probable relation to acute pancreatitis has already been mentioned. In a second fatal case reported by Le Dentu the description of the appearance is insufficient to aid in the solution of the question.

Diagnosis.—The diagnosis of a pancreatic cyst is based upon the presence of a smooth, round globular tumor first appearing in the epigastrium or left hypochondrium, usually separated from the liver and spleen by a resonant zone, gradually increasing in size, slightly movable, especially in a vertical direction. Inflation of the stomach shows that the tumor lies behind and largely below this organ. Aspiration results in the escape, under considerable pressure, of an alkaline fluid, often more or less blood-stained, which usually emulsifies fat, saccharifies starch, more rarely peptonizes albumin. In case of permanent drainage having been established an abundant flow of fluid with similar properties is likely to continue for some time after the operation, and frequently corrodes the edges of the wound.

Mistakes in diagnosis have been frequent. Solid tumors, as sarcoma and cancer, are readily eliminated by the results of puncture. Cystic ovarian tumors are only to be confounded with it when the pancreatic cyst has attained an enormous size. The history then becomes important, as showing the enlargement of the abdomen from below upward in case of the ovarian tumor. The intestines in the latter case are not found in front of the lower portion of the tumor, and the aspirated fluid is free from blood except in the case of a twisted pedicle, which is likely to occur only in the smaller tumors, especially in the dermoid cysts of the ovary. Hydronephrosis of the left kidney is more limited to the left side, extends lower, and the inflated descending colon lies upon it or along its inner edge.

Dropsy of the gall-bladder lies too far to the right, and is directly continuous with the liver.

Aneurism of the abdominal aorta is simulated by the frequently visible and palpable pulsation. The pulsation of the cyst is transmitted, not expansile, and disappears when the patient is placed on the hands and knees.

The most likely confusion in diagnosis is between the pancreatic cyst and a circumscribed collection of fluid in the omental bursa or in the mesentery. The latter may be of pancreatic origin, as an echinococcus-cyst, or may result from dilated lymphatics, as the chylous cyst, or may be due to a peritonitis circumscribed to the omental bursa, the result of traumatism, perforating gastric ulcer, or acute pancreatitis. The composition of the fluid serves to eliminate an echinococcus-cyst, the rare chylangioma, and a suppurative peritonitis. It may be impossible to absolutely exclude a serous or sero-hæmorrhagic peritonitis of the omental bursa, and the treatment is essentially the same.

Prognosis.—The pancreatic cyst may last for years without producing any considerable disturbance. When it attains a considerable size it is always a source of danger, in that it may rupture, compress the stomach and intestines, or interfere with the circulation and respiration. When a dilatation of the duct of Wirsung is the cause, it is possible that diabetes may result, although this event, as well as serious disturbances of the digestive process from a lack of pancreatic juice, are unlikely.

Treatment.—The treatment of the cyst is distinctly surgical, either by the establishment of drainage or by extirpation, the latter operation being preferred when possible. Either operation usually results favorably, although the fistula often remains open for several months.

CANCER OF THE PANCREAS.

Cancer of the pancreas is of chief clinical importance among the neoplastic tumors of this organ. Lymphoma and sarcoma are of rare occurrence, are productive of similar symptoms to those from cancer, while tuberculosis and the syphilitic gumma are anatomical curiosities.

Etiology.—The statistics with reference to the frequency of cancer of the pancreas and its limitation as to size and to the age of occurrence are not based wholly on the primary or secondary occurrence of this lesion. According to Willigk and Lebert, cancer of the pancreas occurs in about 6 per cent. of cancers, and Segré finds that rather more than two-thirds of the patients thus affected are males. It is usually a disease of adult life, being rarely found before the age of thirty years, although a case of cancer of the pancreas has been observed in a child of two years, and the disease may be present at birth. The immediate cause of cancer of the pancreas is unknown, and its association with pancreatic calculi is rare.

Morbid Anatomy.—The anatomical varieties of cancer are the hard, scirrhous, soft, encephaloid or medullary, gelatinous, and cylindrical-celled. The disease is either primary or secondary, and involves any part of the gland, or the entire organ may become diseased. It frequently affects the head, which may be transformed into a mass of the size of a child's head, although smaller tumors are much more common. The softer varieties may be exceedingly vascular, and are at times the occasion of a fatal hæmorrhage into the peritoneal cavity or a serious bleeeding into the duodenum or stomach.

Extension of the disease into adjacent parts is frequent. The neighboring lymphatic glands may become cancerous; nodules may be found in the liver or spleen. The peritoneum may become affected, and adhesions are frequent between the diseased pancreas and the stomach, colon, liver, spleen, gall-bladder, and small intestine. Those portions of the pancreas not cancerous may present no abnormal appearances, or the entire gland may be replaced by cancerous tissue. The frequent limitation of the disease to the head of the pancreas often leads to obstruction or obliteration of the central end of Wirsung's duct or to compression of the common bile-duct. In the latter case the gall-bladder may be found dilated by a colorless fluid.

Symptomatology.—There may be no suggestive symptoms, and cancer be found after death, perhaps sudden, from other causes. As a rule, symptoms of digestive disturbances without apparent cause precede the more characteristic signs. A loss of appetite, perhaps an aversion to meat, a sense of epigastric fulness, belching, nausea, and vomiting are frequent. On the other hand, polyphagia, polydipsia, and polyuria may be present. The vomitus may contain blood, especially in the later stages, from ulceration of the surface of the tumor. The stools may become loose or irregular; they may also contain blood from ulceration, although more constantly quantities of undigested muscular fibre may be found when meat is eaten and there is no diarrhœa: they are rarely fatty. Pain is likely to be complained of, and is, at times, spasmodic, and may be extreme. It is present either in the epigastrium or in the hypochondria, especially in the left, and may extend into the back or around the waist, thus suggesting a lumbar neuralgia. There may be epigastric tenderness as well as pain. The patient is likely to become weak and emaciated, although the loss of flesh and cachexia may be inconsiderable.

A tumor is apparent in one-half of the cases, and may first attract attention after the removal of ascitic fluid. The tumor is deep-seated and fixed at first; as it enlarges it may be somewhat movable, especially in a vertical direction.

The tumor is more or less rounded, slightly lobulated or irregular in shape, usually dense, and is likely to transmit the pulsation of the aorta, and may cause a souffle to be heard, but is without expansile pulsation.

The tumor is the frequent cause of jaundice and ascites, which are, when present, usually among the later symptoms of cancer of the pancreas, although the former may be of early occurrence. The jaundice is the result of pressure upon the common bile-duct, and, according to Segré, occurs in 30 per cent. of the cases. The jaundice may be preceded by chills, and usually persists till death. It may be associated with a tumor from a distended gall-bladder. The ascites is due to the compression or obliteration of the portal vein, and may be accompanied or followed by anasarca and albuminuria. The tumor of the pancreas may be large enough to obstruct the duodenum, in which case persistent vomiting and pain have led to the diagnosis of intestinal obstruction, while dilatation of the stomach is a more frequent result.

There is nothing in the stools characteristic of cancer of the pancreas, although the presence of undigested muscle-fibre is suggestive of a perverted outflow of pancreatic secretion. The urine is apt to be albuminous toward the close of life, but rarely contains sugar, although both glucose and maltose have been found. When the pancreatic secretion does not enter the bowel, the indican is diminished, and salol is not decomposed. Hence the urine is not discolored when this agent is used to test the function of the pancreas.

Course and Duration.—The course of cancer of the pancreas is usually rapid, and the disease may prove fatal within a few months or even weeks, or it may extend over a year or more. Death usually results from progressive emaciation and debility, but may occur suddenly from intraperitoneal hæmorrhage or pulmonary embolism.

Diagnosis.—The diagnosis is dependent upon the presence of the tumor and the evidence furnished by the stools and urine of absent pancreatic secretion. When small the tumor may be found to lie behind the stomach and transverse colon. Inflation of these organs will then be necessary to determine its seat. When large, it may be mistaken for cancer of the pylorus, duodenum, transverse colon or liver.

Cancer of the pylorus is likely to be more freely movable, associated with a dilated stomach, a permanent absence of free hydrochloric acid, while bilious vomiting is unlikely to occur.

It may be impossible to exclude cancer of the duodenum, which is capable of producing the same symptoms. It is probable, however, that most cases of duodenal cancer have extended to the duodenum from the pancreas.

Cancer of the transverse colon is more freely movable, is associated with symptoms of chronic intestinal obstruction, and its relation to the bowel may be determined by rectal inflation. Abundant indican in the urine favors the diagnosis of intestinal cancer and is opposed to that of cancer of the pancreas.

The arrest of pancreatic secretion may be due to causes other than cancer of the pancreas. The most conclusive evidences of this arrest are abundant undigested muscular fibre in constipated stools after a meat diet, and the absence of carbolic acid in the urine when a drachm of salol is taken in divided doses during the day.

Prognosis.—Cancer of the pancreas is inevitably fatal. Death may take place within three months after the occurrence of symptoms. Jaundice and ascites are the symptoms which are of especial prognostic importance. Death may follow the former in four weeks, and the latter in the course of six weeks.

The **treatment** is symptomatic.

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